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STUDY OF ABDOMINAL ADIPOSITY IN TYPE 2 DIABETES MELLITUS AND ITS CORRELATION WITH COMPONENTS OF METABOLIC SYNDROME, ANTHROPOMETRIC PARAMETERS AND INSULIN RESISTANCE IN TYPE 2 DIABETES MELLITUS.

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Dr. Jayesh Patil.

ABBREVIATIONS

ATP III	Adult treatment panel III
BMI	Body mass index
CRP	C-reactive protein
CT	Computed tomography
CVD	Cerebrovascular disease
DKD	Diabetic kidney disease
DR	Diabetic retinopathy
FFA	Free fatty acids
FHS	Framingham heart study
GH	Growth hormone
HDL	High-density lipoprotein
HPA	Hypothalamo-pituitary-adrenal
IDEA	International day for the evaluation of abdominal obesity
IDF	International diabetes federation
IGT	Impaired glucose tolerance
IL-6	Interleukin
MESA	Multi ethnic study of atherosclerosis
MRI	Magnetic resonance imaging
MUHO	Metabolically unhealthy obesity
NAFLDS	Non-alcoholic fatty liver diseases
NCDS	Non-communicable diseases
NIH	National institutes of health

PCOS	Polycystic ovary syndrome
SAT	Subcutaneous adipose tissue
T2DM	Type 2 diabetes mellitus
TNF A	Tumor necrosis factor alpha
VAT	Visceral adipose tissue
VF	Visceral fat
WC	Waist circumference
WHO	World health organization
WHR	Waist/hip ratio

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INTRODUCTION

The type 2 diabetes mellitus prevalence is rapidly increasing, which can be firmly attributed to the modern lifestyle changes that have resulted in increased rates of overweight individuals and obesity. It is estimated that by the year 2030, approximately 366 million people will have diabetes, and more than 90% of them will have T2DM.[1] Along with obesity and physical inactivity, there is a significant risk of developing insulin resistance. Several facts correlate with the phenomena, as insulin resistance is observed in approximately 90% of patients with T2DM and 66% of individuals with impaired glucose tolerance (IGT). The worst part of insulin resistance is that it doubles the risk for cardiovascular disease, which is the ultimate cause of death in about 80% of patients with T2DM.[2] This fact suggests that an essential part of the higher cardiovascular risk observed is due to its proatherogenic effects.

Central (intra-abdominal) obesity is observed in the majority of patients with T2DM, which is associated with insulin resistance, mainly at the level of adipose tissue, liver, and skeletal muscle. To assess the weight-related health risk, BMI and waist circumference (WC) are the two recommended measurements. BMI has gained international acceptance because of its associations with adiposity, disease risk, and mortality. At the same time, waist circumference is associated with visceral adipose tissue, insulin resistance, metabolic syndrome, and diabetes mellitus type 2.[3-5] BMI, however, may not accurately reflect health risks among older adults who may have a BMI value in the healthy range despite muscle loss and excess abdominal fat. Hence, Waist circumference and body composition assessments are likely to provide more valuable information regarding health risks. Epidemiological studies have shown that South Asians also are more likely to have central obesity, increased waist/hip ratio (WHR), and glucose intolerance compared with Caucasians.[6,7] However, data related to increased WHR and the prevalence of diabetes in Asian Indians are controversial, and detailed studies of body fat distribution with insulin resistance have not been reported.[8] Moreover, data suggest that the proposed cutoffs for defining overweight and obesity are not relevant for Asian Indians and that Asian Indians are endangered of emerging obesity-related comorbid conditons at lesser grade of body mass index (BMI) and waist circumference (WC).[9]

Adipose tissue dysfunction is characterized by ectopic fat deposition in the abdominal viscera and liver, inflammatory and adipokine dysregulation, and insulin resistance and maybe a more important mediator of diabetes development than total fat mass in obese individuals.[10,11] Earlier studies on Asian Indians have reported an association of waist

circumference with diabetes, suggesting that increased fat accumulation in the abdominal cavity may be one of the contributors to diabetes in this ethnic group. Waist circumference showed a strong correlation with visceral and central abdominal fat in both diabetic and nondiabetic subjects.[12,13] However, BMI, which correlated with central abdominal fat in both diabetic and nondiabetic subgroups, failed to show an association with visceral fat in the diabetic group, suggesting that BMI is not as good as waist circumference in predicting visceral adiposity in diabetic subjects. Therefore, the primary aim of the current research was to examine the relative contributions of abdominal adiposity and its correlation with components of metabolic syndrome, anthropometric parameters, and insulin resistance among diabetic men and women. Magnetic resonance imaging (MRI) and Computed Tomography (CT) can accurately quantify the volumes of visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) and shows good accuracy with ultrasonography.[14] But its cost and need for specialized personnel limit their use. Ultrasound is a useful assessment tool when other options aren't available because it is a portable imaging technology that can quickly evaluate regional body composition. Hence, we also equiped the sonography techniques to measure abdominal adiposity.

AIM & OBJECTIVES

Aim:

To study abdominal adiposity and to correlate it with components of Metabolic Syndrome, Anthropometric Parameters and Insulin Resistance in patients with type 2 diabetes mellitus.

Objective:

- To estimate abdominal adiposity by Ultrasound.
- To correlate adiposity with BMI.
- To correlate component of metabolic syndrome (Blood pressure, Triglycerides, WC, abnormal fasting blood sugar and Insulin resistance) with BMI in diabetics.
- To correlate abdominal adiposity with Insulin resistance.

Research Question: Is there any association between abdominal adiposity and with components of Metabolic Syndrome, Anthropometric Parameters and Insulin Resistance in patients with type 2 diabetes mellitus ?

REVIEW OF LITERATURE

Diabetes mellitus type 2 is a chronic, fatal condition that causes macro- and micro-vascular complications like cardiovascular and cerebrovascular disease (CVD), diabetic kidney disease (DKD), and diabetic retinopathy (DR), all of which are linked to increased disability, a decline in quality of life, and a shorter life expectancy.[15] A marked increase in the prevalence of overweight and obesity has contributed to a doubling in type 2 diabetes mellitus incidence over the past three decades. Increasing rates of diabetes among obese individuals have counterbalanced reductions in other cardiovascular disease (CVD) risk factors and are the primary factor contributing to a slowed decline in CVD event rates in the general population. The purpose of the present research is to bring together available information on the distribution and determinants of abdominal adiposity and insulin resistance through a comprehensive review of published research worldwide. The study provides an understanding of the subject, with part 1 focusing on the difference in cutoffs of obesity and abdominal obesity for Asian Indians, followed by factors that put diabetic obese at high risk for atherosclerosis and thrombosis, as well as reviewing and comparing the studies that was ultimately help us in the discussion chapter of this dissertation.

Type 2 Diabetes Mellitus (T2DM):

The development of Diabetes Mellitus (T2DM), one of the most predominant metabolic illnesses in the world, is primarily attributed to a confluence of two key factors: the incapability of insulin-sensitive tissues to respond to insulin and defective insulin secretion by pancreatic beta-cells.[16]

Insulin release and action have to meet the metabolic demand precisely; Therefore, it is essential to closely manage the molecular processes involved in insulin synthesis, release, and tissue response. As a result, flaws in any of the pathways can result in a metabolic imbalance and T2DM pathogenesis.

The greatest significant modifiable risk factor for type 2 diabetes is excess weight, because 85–90% of population with type 2 diabetes are either overweight or obese.[17]

The risk factors of the T2DM from a preventive, treatment, or interventional are:

- Modifiable
- Non-modifiable.

The risk of an individual developing T2DM depends on the presence and combination of exposure to modifiable and non-modifiable risk factors. Non-modifiable risk factors like age, ethnicity and family history may alert individuals of their baseline risk of contracting the

disease. Modifiable risk factors like obesity, harmful use of alcohol, physical inactivity, and dietary factors can be altered to prevent the disease.[18]

Obesity:

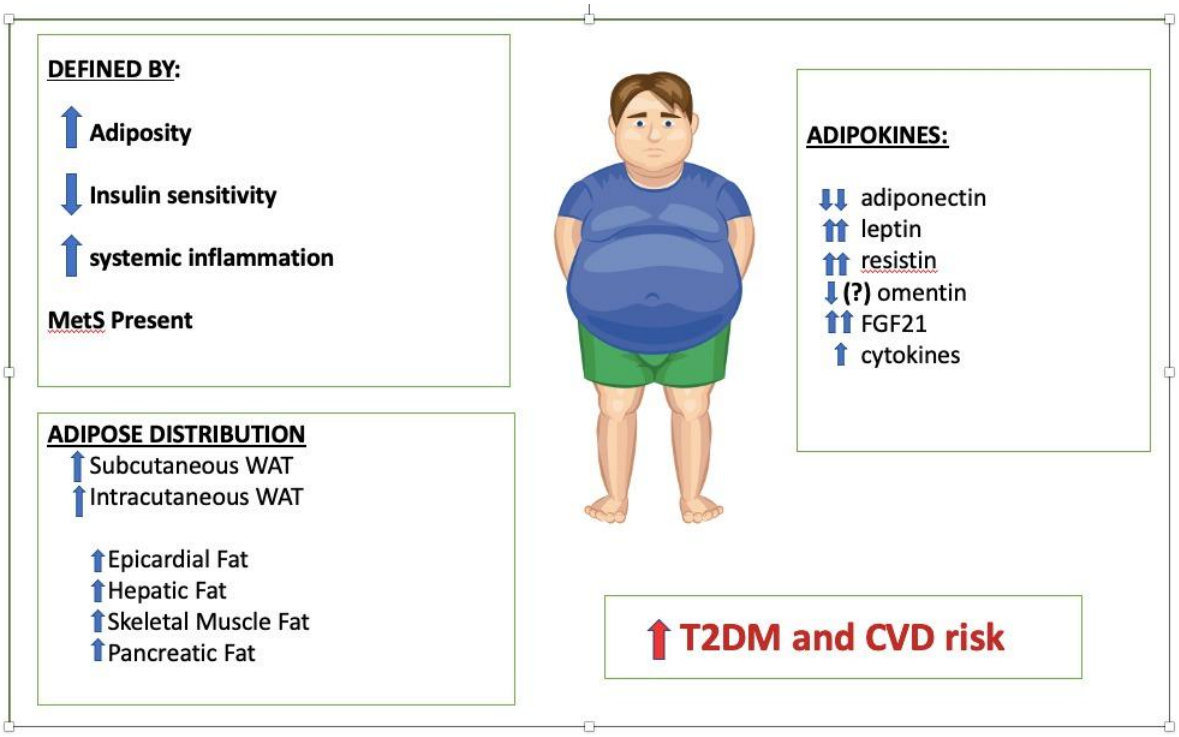


Figure 1: Metabolically unhealthy obesity (MUHO).

Obesity is characterized by excessive body fat accumulation and storage; however, clinically, exact evaluation of extra body fat is tough to execute. Body weight can give an indirect measure of fat stores, but the variability of body builds, and composition makes it hard to define a specific level for obesity. The BMI is the most often used indicator and prognostic indicator of obesity.[19]

Pathophysiology:

The mechanisms leading from obesity to type 2 diabetes are complicated and not fully elucidated. It seems that free fatty acids and adipokines produced by the abdominal adipose tissue decrease insulin sensitivity and increase insulin demands.[21] The classical perception of adipose tissue as a storage place of fatty acids has been replaced by the notion that adipose tissue has a central role in lipid and glucose metabolism, and produces a large number of hormones and cytokines (e.g. tumor necrosis factor-alpha, interleukin-6, adiponectin, leptin and plasminogen activator inhibitor-1).[22] Therefore, the increased prevalence of excessive visceral obesity and obesity-related cardiovascular risk factors is closely associated with the

rising incidence of type 2 diabetes. Genes concerned with carbohydrate, lipid and amino acid metabolic pathways, neuronal function and inflammation play a significant role in the pathobiology of obesity and type 2 diabetes.[23] Increased oxidative stress together with decreased anti-oxidative defense seem to contribute to decreased insulin sensitivity and impaired insulin secretion response in obese diabetics, and may be hypothesized to favor the development of diabetes in obese.[24]

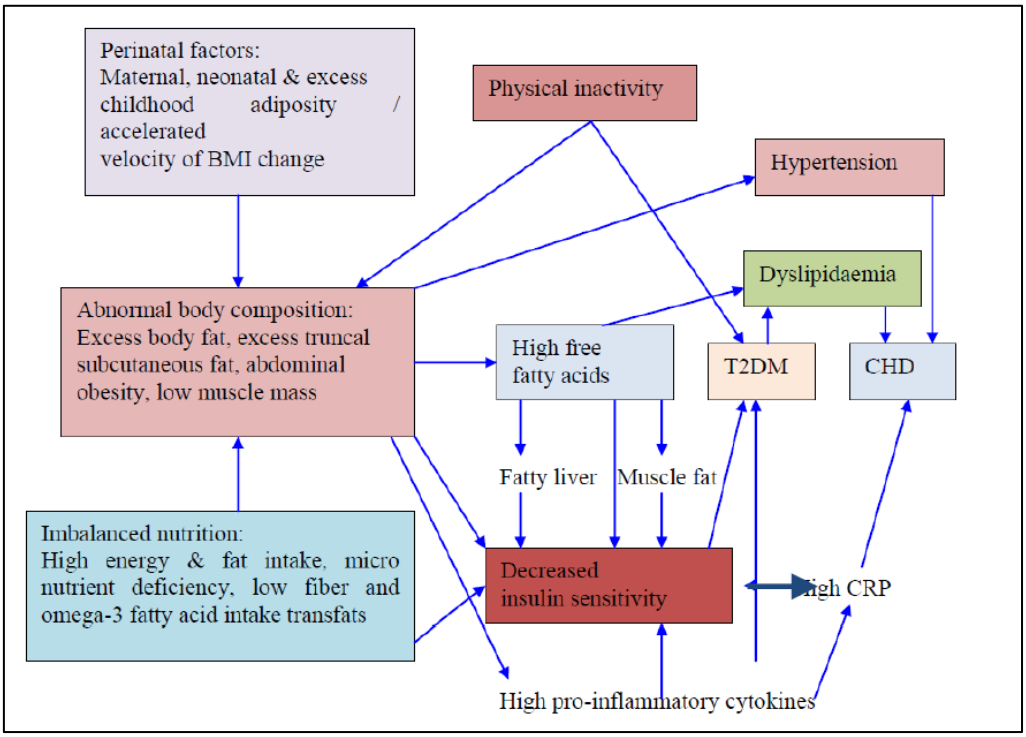

Perinatal factors:
Maternal, neonatal & excess childhood adiposity / accelerated velocity of BMI change
Physical inactivity
Hypertension
Dyslipidaemia
Abnormal body composition:
Excess body fat, excess truncal subcutaneous fat, abdominal obesity, low muscle mass
High free fatty acids
T2DM
CHD
Fatty liver
Muscle fat
Imbalanced nutrition:
High energy & fat intake, micro nutrient deficiency, low fiber and omega-3 fatty acid intake transfats
Decreased insulin sensitivity
High CRP
High pro-inflammatory cytokines

Figure 2: Complex interactions in development of insulin resistance, type-2 diabetes and coronary heart disease in South Asians.[25]

Table 1: Cut-offs of obesity and abdominal obesity for Asian Indians *vs.* international criteria.[26]

Variable	Consensus guidelines for Asian Indians.[a]	Prevalent International Criteria
Generalized obesity	Normal: 18.0–22.9	Normal: 18.5–24.9 b
(BMI cut-offs in kg/m2)	Overweight: 23.0–24.9 Obesity: >25	Overweight: 25.0–29.9 [b] Obesity: >30 b
Abdominal obesity (Waist circumference cut-offs in cm)	Men: >90 c Women: >80 [c]	Men: >102 d Women: >88 [d]

Notes: a- From Consensus guidelines for Asian Indians; b- According to World Health Organization guidelines; c- Both as per Consensus Guidelines for Asian Indians and International Diabetes Federation; d- According to Modified National Cholesterol Education Program, Adult Treatment Panel III guidelines.

Etiology of visceral obesity:

Age:

Men and postmenopausal women, who, on average, have up to twice as much visceral adipose tissue as premenopausal women, are at particular risk for increased visceral adipose tissue deposition with age. However, visceral adipose tissue deposition was higher in middle-aged premenopausal women than in young women, demonstrating that ageing was linked to an increase in abdominal adiposity in women even before the start of menopause.[27]

Gender:

Human males and females differ significantly in terms of body fat distribution. Such notable anatomical differences attributable to regional adipose tissue partitioning are unique to this species. Men are more likely to store adipose tissue in the upper body (trunk, abdomen), whereas women usually store adipose tissue in the lower body (hips, thighs).[28]

Sex hormones:

The marked sex dimorphism in the human body fat patterning indirectly suggests that sex hormones play a crucial role in regional fat accumulation.[29] Researchers frequently come to the conclusion that abdominal obesity and hyperinsulinemia in women are caused by hyperandrogenism based on the frequent observation of abdominal obesity in patients with polycystic ovarian syndrome (PCOS). Low circulating levels of total testosterone in males are typically linked to visceral and abdominal obesity.[30,31] Due to methodological restrictions in measuring free testosterone, it is still unclear if free testosterone levels are associated with body fat distribution patterns.

Genetics:

Numerous studies on the genetic factors contributing to obesity have been conducted during the last few decades. According to family research, there is a 50% heredity rate for total body fat mass.[32]

Ethnicity:

The pronounced differences in regional adipose tissue distribution among various populations worldwide are well known. For a given weight gain, some populations may be prone to accumulate adipose tissue in the subcutaneous adipose depots. In contrast, other populations may be more likely to collect adipose tissue in the visceral cavity. Ethnicity, therefore, critically needs to be considered in identifying high-risk cases of obesity, especially in defining cutoff values of anthropometric measurements.[33]

Growth hormone:

Human studies have shown that growth hormone (GH) levels or secretion are altered in visceral obese individuals with a high cardio-metabolic risk. Many reports suggested that the association between visceral and/or abdominal obesity is independent of total adiposity.[34,35] Adiposity and fat distribution, along with aging and the sex steroid milieu, interact in a highly complex manner to modulate GH secretion.[36]

Hypothalamic-Pituitary-adrenal axis stress and Glucocorticoids:

Excessive circulating glucocorticoid concentrations, as observed in Cushing's syndrome, create a pathological phenotype of abdominal obesity, dyslipidemia, insulin resistance, and hypertension. In most cases, cortisol hyper-secretion origins from the pituitary gland (Cushing's disease) and results from excessive adrenocorticotropic hormone secretion. In comparison, individuals with idiopathic abdominal obesity share several morphological and

metabolic alterations observed in Cushing's syndrome, alterations in the sensitivity and drive of the hypothalamic-pituitary-adrenal (HPA) axis are much subtler.[37]

Sedentary Lifestyle/Physical activity:

A sedentary lifestyle could make an individual more susceptible to positive energy imbalance when facing a contemporary diet rich in energy-dense, processed foods, and sugar-containing beverages. Not firmly established whether a lack of physical activity increases susceptibility to selective abdominal fat deposition.[38]

Metabolic risk is determined by fat distribution:

The location of extra fat is crucial in terms of metabolic risk. Epidemiological and metabolic research have provided evidence that the area of extra fat rather than its total amount is more directly related to the detrimental metabolic effects of excess fat. In actuality, central fat accumulation rather than overall fat mass may be a stronger predictor of elevated risk for type 2 DM and CVD.[39,40]

Abdominal obesity:

AO, also known as central adiposity, visceral, android, or male-type obesity, is considered to be a harmful type of obesity with serious consequences. It is defined as an excess of intra-abdominal fat. It is a common health condition seen in South Asians, is positively related to non-communicable diseases (NCDs), and has seen a remarkable increase over the years.[41] AO is an indicator of the accumulation of triacylglycerols in the liver and muscles. It has been strongly linked to given common NCDs, in particular: cardiovascular diseases (CVDs), diabetes, hypertension, cancer, kidney diseases, and non-alcoholic fatty liver diseases (NAFLD).[42,43]

Anthropometric measurements:

Body mass index (BMI):

BMI is a general measure of obesity and has reportedly been linked to a higher incidence of both type 2 diabetes and high blood pressure.[44] BMI is measured as weight (in kilograms) divided by the height (in meters) squared. Both the World Health Organization (WHO) task force and the National Institutes of Health (NIH) guidelines stipulate normal, overweight, and obesity cutoffs according to BMI. Although there are some restrictions, BMI generally correlates with body fat percentage.

Because the cutoffs underestimate risk in the Asian population, the WHO and NIH guidelines for Asian individuals define overweight as a BMI between 23 and 24.9 kg/m2 and obesity as a BMI >25 kg/m2.[45]

Table 2: Obesity classification according to WHO and ESI Clinical Practice Guidelines.[46]

Category	WHO (BMI)	Asian criteria (BMI)
Underweight	<18.5 kg/m^2	<18.5 kg/m^2
Normal	18.5–24.9 kg/m^2	18.5–22.9 kg/m^2
Overweight	25–29.9 kg/m^2	23–24.9 kg/m^2
Obese Type 1	30-40 kg/m^2	25 – 29.9 kg/m^2
Obese Type 2 (morbid obese)	40.1-50 kg/m^2	30-34.9 kg/m^2
Obese Type 3 (super obese)	>50 kg/m^2	>35 kg/m^2

There has been a rise in the prevalence of DM and mortality with increasing BMI in patients within the normal range. This trend may be explained by the pattern of fat distribution, with a more central and particularly visceral fat accumulation being associated with increased risk for DM and cardiovascular disease; This facilitates the assessment of health risks in people with a BMI between 25 and 35 kg/m2. Recent data failed to show an increase in mortality in overweight individuals compared with individuals of average weight, possibly due to improved cardiovascular risk management. In contrast, obese and underweight individuals had higher mortality rates. The association between increased gluteofemoral adipose tissue and cardiovascular risk is much weaker. Another pitfall of relying on BMI alone is that muscular patients may have relatively high BMIs but low-fat mass. In the elderly, BMI may underestimate fat mass due to a decrease in lean body mass. The third drawback is that because body fat percentage changes with age, sex, and ethnicity, BMI may not be a reliable indicator of obesity in some populations.[46] The BMI has another drawback in that it does not

record information about where body fat is located. This is an essential variable in assessing the metabolic and mortality consequences of excessive fat accumulation.[42]

Waist circumference:

A different anthropometric measure, waist circumference is a straightforward, conventional, and clinically applicable way to measure abdominal adiposity.[47] WC reflects the magnitude of the abdominal fat tissue, including subcutaneous and visceral adipose tissue. Although it is a simple and crude measurement, it correlates with VAT and, when increased, has been shown to confer an elevated risk for DM, CVD, and mortality, independent of BMI. Although it is a simple and crude measurement, it correlates with VAT and, when increased, has been shown to confer an elevated risk for DM, CVD, and mortality, independent of BMI.[48]

Measurement of WC:

Different anatomic landmarks used to measure WC include the midpoint between the lowest rib and the iliac crest, the umbilicus, the narrowest or widest WC, just below the lowest rib, and just above the iliac crest. The NHLBI practical guide recommends measuring just above the iliac crest. WC measurement should be done with the patient standing, bare midriff, after exhaling, with both feet touching and arms hanging freely. A material that cannot be easily stretched should be used to make the measuring tape.; it should be placed vertically on the long axis of the body and horizontally on the floor and applied with tension, but it should not put pressure on the abdominal wall.[49]

Of note, additional guidance provides different methods to quantify waist circumference. For example:

- WHO recommends that the measurement is made at the approximate midpoint between the lower margin of the last palpable rib and the top of the iliac crest.[50]
- The NIH and NHANES III protocol recommend the superior aspect of the iliac crest as the site of measurement.[51]
- The NIH Multi Ethnic Study of Atherosclerosis (MESA) protocol measures waist circumference at the level of the umbilicus or navel.

Additionally, each measurement needs to be taken twice , and the average should be calculated if the interpretations are within 1 cm of one another. Repeat the two measurements if the discrepancy between the two values is greater than 1 cm. Different standards also have different cutoff points for what constitutes abdominal obesity. According to the International Diabetes Federation (IDF), central obesity is defined as a waist circumference of more than

94 cm for males and more than 80 cm for women who are Europid. The IDF guidance also recommends ethnic-specific cutoff values, for example, a waist circumference greater than 90 cm for south Asian, Chinese, and Japanese men.[52] The Adult Treatment Panel III (ATP III) uses a cutoff value of higher than 102 cm for men and higher than 88 cm for women. NICE classifies three risk groups. For men, a waist circumference less than 94 cm is low, 94–102 cm is high, and greater than 102 cm is very high. For women, a waist circumference less than 80 cm is low, 80–88 cm is high, and greater than 88 cm is very high.[52]

In the International Day for the Evaluation of Abdominal Obesity (IDEA) study, which included 69,409 subjects from 63 countries who consulted a primary care physician, BMI and particularly WC were strongly linked to DM.[53]

Waist-to-hip ratio:

Waist-to-hip ratio (WHR) is another commonly used anthropometric measure to identify central obesity. Hip circumference was recorded around the most comprehensive portion of the buttocks. The WHO guidance defines central obesity if a waist-hip ratio greater than 0.90 in males and greater than 0.85 in females. A high WHR has been associated with increased cardiovascular and DM risk in epidemiological studies.[54]

Limitations:

Using a ratio measure instead of a single measurement is more prone to interobserver variations and calculation errors. Since WHR is a ratio measure, there is a possibility that both lean individuals and obese individuals would have the same ratio.[45]

Measurement of Abdominal adiposity:

Ultrasonography (US) is an alternative, noninvasive, reliable method to estimate these two fat compartments. Compared to MRI or computed tomography, its validity has been tested in adult women, obese adults, and diabetic adults; however, it is essential to test its validity in older individuals as fat accumulation in the abdomen increases with age.[55,56]

Abdominal obesity, as a clinical feature of excessive visceral fat accumulation, is usually associated with a cluster of cardiovascular risk factors, defined by the WHO as ‘metabolic syndrome.[57]

Earlier studies on Asian Indians have reported an association of waist circumference with diabetes, suggesting that increased fat accumulation in the abdominal cavity may be one of the contributors to diabetes in this ethnic group. Waist circumference strongly correlated with visceral and central abdominal fat in diabetic and nondiabetic subjects. However, BMI,

coupled with central abdominal fat in both diabetic and nondiabetic subgroups, failed to show an association with visceral fat in the diabetic group, suggesting that BMI is not as good as waist circumference in predicting visceral adiposity in diabetic subjects. However, there is evidence that certain visceral fat distribution patterns rather than subcutaneous fat distribution around the waist may be associated with higher metabolic risk. Recent research have shown that visceral adipose tissue (VAT) has a different and more detrimental metabolic role than subcutaneous adipose tissue, covering populations of diverse genders, ages, BMI levels, and races (SAT).[58,59] The relationship between the distribution of abdominal fat as determined by CT and several metabolic risk factors was investigated cross-sectionally as part of the Framingham Heart Study (FHS). Both SAT and VAT were strongly correlated with fasting plasma glucose and HOMA insulin resistance in a sizable community-based, mostly white sample. The associations were more substantial with VAT than with SAT. VAT but not SAT contributed significantly to risk factor variation after adjustment for BMI and waist circumference.[11,59]

Similarly, in a study by **Philipsen et al.**, after adjustment for waist circumference and body fat percentage, associations between VAT and glucose outcomes are still significant for all results.[60] In a study of Mexican Americans, **Wei et al.** found that WC was the best obesity-related predictor of non-insulin-dependent diabetes, with a predictive effect equal to that of WHR and BMI combined. The authors argue that the abdominal localization of body fat is a more important determinant than the population's total body fat.[61]

Metabolic syndrome:

This syndrome is characterized by abdominal obesity, insulin resistance, dyslipidemia (high triglyceride level and low high-density lipoprotein [HDL] cholesterol level), hypertension, high fasting glucose, procoagulant (elevated plasminogen activator inhibitor [PAI-1] levels) and pro-inflammatory state, i.e., elevated tumor necrosis factor (TNF), C-reactive protein (CRP) and interleukin (IL)-6.[57]

Table 3: Definitions of metabolic syndrome.[62]

Variable	NCEP ATP III	WHO	EGIR	IDF
Absolutely required	None	Insulin resistance (IGT, IFG, T2D or other evidence of IR)	Hyperinsulinemia (plasma insulin >75th percentile)	Central obesity (waist circumference§): 94 cm (M), ≥ 80 cm (F)
Criteria	Any three of the five criteria below	Insulin resistance or diabetes, plus two of the five criteria below	Hyperinsulinemia, plus two of the four criteria below	Obesity, plus two of the four criteria below
Obesity	Waist circumference: >40 inches (M), >35 inches (F)	Waist/hip ratio: >0.90 (M), >0.85 (F); or BMI >30 kg/m2	Waist circumference: ≥94 cm (M), ≥ 80cm (F)	Central obesity already required
Dyslipidemia	TG 150 mg/dl or Rx	TG 150 mg/dl or HDL-C:	TG 177 mg/dl or HDL-C	TG 150 mg/dl or Rx
Hypertension	>130 mmHg systolic or >85 mmHg diastolic or Rx	≥140/90 mmHg	≥140/90 mmHg or Rx	>130 mmHg systolic or >85 mmHg diastolic or Rx

The association between MetS and T2DM is probably because the definition of MetS includes elevated FPG, a powerful predictor of future T2DM. The risk of developing T2DM increased with the number of MetS components.[63] FPG was the most robust individual predictor. The most potent combination of two elements that excluded elevated FPG for T2DM risk was the combination of elevated triglycerides and reduced HDL cholesterol. Moreover, the combination of both components was associated with an increased risk of T2DM comparable with elevated FPG. The highest risk combination of the three components

was that combining elevated FPG, elevated triglycerides, and reduced HDL cholesterol.[64] The combination of elevated triglyceride and reduced HDL cholesterol, commonly named atherogenic dyslipidemia, is associated with T2DM. High triglyceride level, accompanied by a low HDL cholesterol level, has consistently been shown to be a surrogate marker of insulin resistance which is a solid predisposing condition for T2DM. Hypertriglyceridemia directly relates to insulin resistance and hyperglycemia, and the origin of low HDL cholesterol is closely linked to hypertriglyceridemia. Thus, elevated triglyceride-rich lipoproteins produce low HDL cholesterol via exchange processes between these lipoproteins and HDL cholesterol. However, the combination of both components was associated with a synergistically increased risk of developing T2DM. Although it is evident that insulin resistance is closely related to elevated triglyceride levels accompanied by low HDL cholesterol, the cause is unclear. Dyslipidemia has been associated with the risk of developing T2DM.[65]

Pathophysiology:

There is no consensus that different METS elements form distinct pathologies or fall under a typical, broader pathogenic process. METS is a result of how genetic and environmental factors interact.

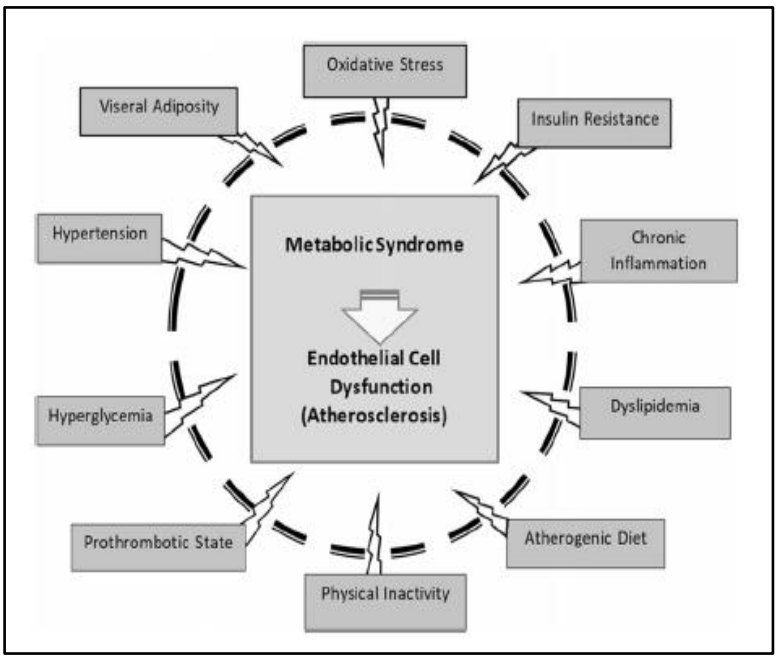


Figure 3: Schematic presentation of the metabolic syndrome components that affect the endothelium and the consequences of endothelial dysfunction.[66]

The main factor which is responsible for the development of cascade of events for the development of METS is visceral obesity. Insulin resistance, chronic inflammation and neurohormonal activation helps in the progression of METS and leads to the development of CVDs and T2DM.Visceral fat accumulation results in fat redistribution and causes isceral adiposity. This visceral adiposity results in the development of insulin resistance.[67]

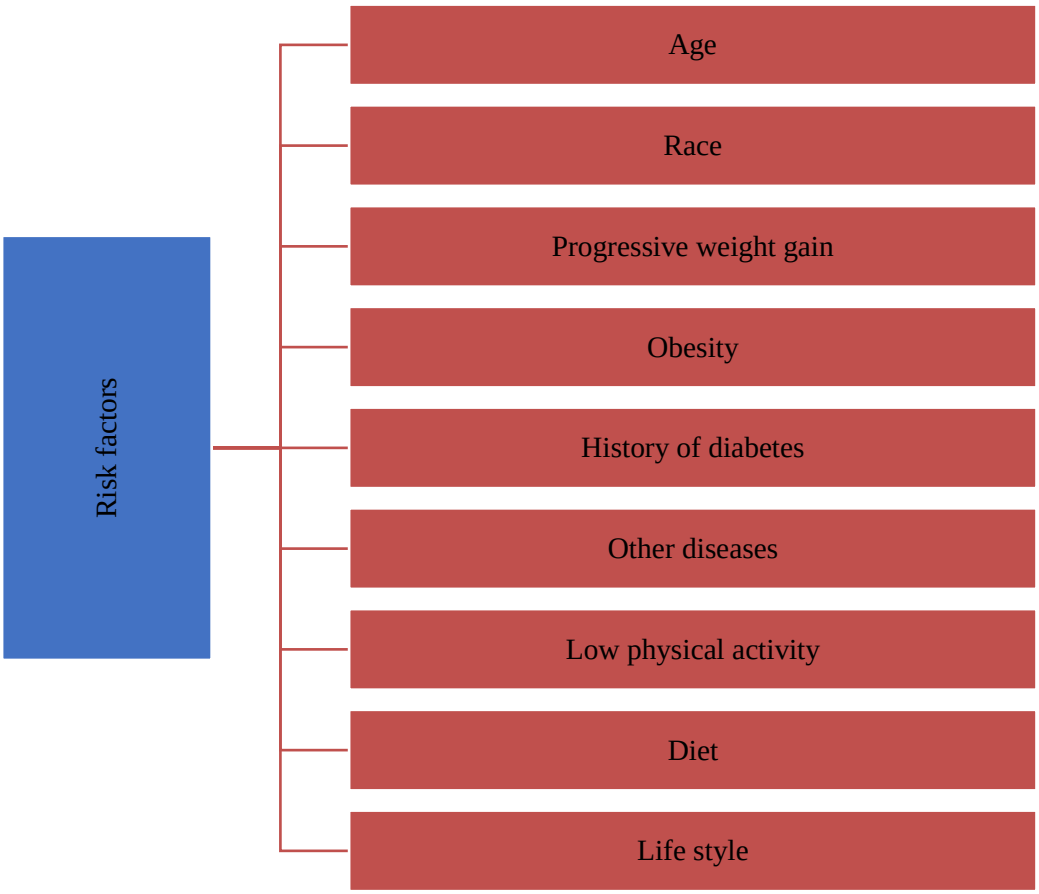


Figure 4: Risk factors for the development of MetS.

METS is associated with increased levels of free fatty acids and accumulation of cell fat in the liver and skeletal muscles. This accumulated cell fat results in defects in glycogen synthesis and glucose metabolism. As the VAT deposits increase, the adipocyte function is further reduced and causes macrophage infiltration. Then adipocytokines releases and aggravate the inflammatory insulin-resistant environment.[68] The various adipocytokines released are tumor necrosis factor-alpha (TNF α), adiponectin, resistin, and lipase individually modulate insulin resistance, lipid metabolism, and inflammatory responses. All these acts are in concordance and promote the direct pro-atherogenic vascular effects.[57] Increased flux of free fatty acids (FFA) from the periphery to the liver in the Insulin resistant state also stimulates the synthesis of hepatic TG. This promotes the assembly and secretion of

TG-containing VLDL and increases TG levels. This results in decreased LDL levels. Thus, activation of innate immunity and adipokines are the underpinned mechanisms for the generation of insulin resistance and atherogenic dyslipidemia.[69]

Insulin resistance:

Insulin resistance is one of the vital pathophysiological processes in developing Type 2 diabetes and is strongly associated with visceral adiposity.[70] Numerous theories have been put up to explain the link between insulin resistance and obesity. In summary, these include:

- Hepatic lipid excess: In obesity, the capacity to store lipids in adipose cells becomes limited. These excess lipids in free fatty acids pass into the liver via the portal circulation, leading to increased hepatic insulin resistance and hepatic gluconeogenesis. This phenomenon also leads to impaired beta-cell insulin secretion and insulin resistance within skeletal muscle. The result is hyperglycemia due to reduced peripheral glucose uptake;
- Adipocytes are metabolically active cells, secreting many adipokines, such as leptin, adiponectin, and resistin. Dysfunctional adipokine regulation due to obesity leads to increased hepatic glucose production, reduced skeletal muscle glucose utilization, increased food intake, and reduced energy expenditure;
- Adipocytes secrete adipokines and pro-inflammatory cytokines such as TNF-α and IL6 that are involved in developing insulin resistance.[45]

Insulin resistance & T2DM:

Type 2 diabetes is the most common result of insulin resistance (T2DM). T2DM is estimated to develop 10 to 15 years before insulin resistance. Typically, an increase in endogenous insulin production occurs as a result of insulin resistance. Increased endogenous Insulin levels, an anabolic hormone linked to insulin resistance, cause weight growth, which in turn makes insulin resistance worse.[71,72] This vicious cycle continues until there is hyperglycemia due to insufficient pancreatic beta-cell activity to satisfy the increased demand for insulin brought on by insulin resistance. Glycemic levels increase to levels associated with T2DM as a result of a persistent discrepancy between insulin demand and insulin output. In addition to T2DM, the disease associated with insulin resistance includes obesity, cardiovascular disease, nonalcoholic fatty liver disease, metabolic syndrome, and polycystic ovary syndrome (PCOS).[73]

Table 4: Reviewing the studies of Correlation Between Adiposity Indexes and Homeostatic Model Assessment for Insulin Resistance.[74]

Index	Study Population	Correlation Coefficient	Source
Intra-abdominal fat thickness	Diabetic men	0.43	Kim et al.
	Diabetic women	0.30	Kim et al.
	Obese women	0.27	Ribeiro-Filho et al.
	Obese women	0.27	Faria et al.,
Abdominal wall fat	Men in general population	–0.07	Soyama et al.
	Diabetic men	Non-significant	Kim et al.
	Diabetic women	Non-significant	Kim et al.
Maximum preperitoneal fat thickness	Men in general population	0.11	Soyama et al.
	Diabetic men	Non-significant	Kim et al.,
	Diabetic women	Non-significant	Kim et al.
Maximum preperitoneal fat thickness	Healthy men	0.69	Liu et al.
	Healthy women	0.56	Liu et al.
	Healthy volunteers	0.32	Liu et al.

Index	Study Population	Correlation Coefficient	Source
Minimum subcutaneous fat thickness	Men in general population	0.18	Soyama et al.
Mesenteric fat thickness	Healthy men	0.77	Liu et al.
	Healthy women	0.58	Liu et al.
	Healthy volunteers	0.39	Liu et al.
Preperitoneal circumference	Healthy volunteers	0.43	Merino-Ibarra et al.

Complications: T2DM patients are more vulnerable to different forms of both short- and long-term complications. People with type 2 diabetes mellitus are at higher risk of many difficulties, mainly due to complex and interconnected mechanisms such as hyperglycemia, insulin resistance, low-grade inflammation, and accelerated atherogenesis. The complications include:

- Macrovascular diseases (hypertension, hyperlipidemia, heart attacks, coronary artery disease, strokes, cerebral vascular disease, and peripheral vascular disease).
- Microvascular diseases (retinopathy, nephropathy, and neuropathy).
- Cancers.[73]

REVIEW OF RELATED LITERATURE

Awasthi A et al.[74] in 2017 compared the correlation between type 2 diabetes mellitus and different anthropometric measurements. This was a case-control study and recruited 102 individuals for the study. The proportion of cases with Body Mass Index (BMI) ≥ 25 kg/m2 was 55% as compared to 22% of controls, and this association was statistically significant ($p < 0.05$). The proportion of cases with high waist circumference cutoffs (WC) was 74.5% as compared to 45.1% of healthy individuals, and this association was also statistically significant ($p < 0.05$, OR = 3.56). A Receiver Operating Characteristic (ROC) curve for both genders revealed the highest area under the curve for body mass index (area = 0.787). Body mass index had the best discriminatory power. The proportion of cases with waist to hip ratio (WHR) high was 84.2% when compared to 80.4% of healthy individuals, and this association was not statistically significant. (P=0.799). Waist to stature ratio was not a sensitive marker, especially for females. The majority of the patients in the cases group, 88.2%, had abnormal WHR compared to the 64.7% of individuals with normal WHR, and the difference was statistically significant. (P=0.005). A strong association between obesity indices and diabetes were identified. BMI and WC could be used in clinical practice to suggest lifestyle modification.

Qureshi SS et al.[75] in 2019 compared the body mass index (BMI) and waist-to-hip ratio (WHR) in their ability to predict type 2 diabetes risk. The study included 804 diabetics and 396 nondiabetics aged between 30 and 60. 9.1% of nondiabetics had normal WHR compared to 2.1% of diabetics, whereas 90.9% of nondiabetics had increased WHR and 97.9% of diabetics had increased WHR, overall there is a strong association between diabetic patients with an increased waist-hip ratio (P-value <0.001). In the diabetic group, 12 (1.5%) were underweight, 75 (9.3%) had a normal BMI, 85 (10.6%) were overweight, and 632 (78.6%) were obese. There was a significant association between diabetes and raised BMI (p-value <0.001). Both BMI & WHR were further compared using the ROC curve, which found that WHR had an area under ROC of 0.720 & BMI had 0.680, suggesting that WHR is a better predictor of diabetes than BMI. BMI and WHR were strong discriminators of T2DM, but WHR was found superior according to ROC value.

Biadgo B et al.[76] in 2018 studied the prevalence of MetS and its components among T2DM patients. This was a cross-sectional study, and 159 participants were enrolled. 119 (59.7%) were females with a mean age of (49.8±8.7) years. The prevalence of MetS was

66.7% in NCEP-ATP III and 53.5% in IDF definitions. The frequencies of MetS components were the highest for elevated TG (56.6%) and elevated blood pressure (55.4%) using NCEP-ATP III, central obesity (61.0%), and elevated TG (62.3%) in the IDF criteria. The regression analysis showed that increased age, being female, having high BMI, having diabetes for over five years, and having poor glycemic control were significantly associated with metabolic syndrome. The author concluded that the prevalence of MetS and its components among T2DM patients was high.

Akhter O et al.[77] in 2015 undertook a study to evaluate the body total and central fat percentage in type 2 Diabetes Mellitus patients and to check the correlation with BMI, waist circumference, and metabolic profile. One hundred and seventy-five patients (95 males and 80 females) with a mean age of 54.1 ± 12 years were evaluated. The mean duration of diabetes was 8.1 years, the mean HbA1c was 8.1%, 53.7% were on oral agents, and the rest were on Insulin with or without oral agents. Hypertension was present in 65.7%, 13.7% had known coronary artery disease, and 2.3% had cerebrovascular disease. The mean BMI in males was 29.1 ± 4.74 kg/m2, and in females, 31.7 ± 5.3 kg/m2. The mean waist circumference in males was 107.3 ± 16.6 cm and 103 ± 12 cm in females. The total body fat percentage (%BF) in males was 30.9 ± 7.1%. 40 ± 8.2%, with 89% of the entire cohort having total body fat percentage above the normal, less than 25% central fat percentage was 13.3 ± 5.2% in males and 14.6 ± 5.5% in females, with 79.4% of the cohort having increased central fat (average <9%). Total and central body fat correlated with BMI (r = 0.68, P < 0.001) and waist circumference (r = 0.66, P < 0.001) but not with HbA1c, triglyceride level, fasting, or random blood glucose levels. Women had significantly higher total body fat percentage than men (P < 0.001), although the central fat percentage was similar in both sexes. Increased body fat percentage and waist circumference are seen, especially in women, and central body fat percentage was identical in both sexes among patients with type 2 diabetes mellitus.

M. Premanath et al.[78] in 2014 conducted a study to measure Visceral Fat (VF) and Subcutaneous Fat (SCF) by Ultrasound in obese and non- obese diabetics. This was a cross-sectional observational study, and 160 subjects were recruited. All subjects were recruited in four groups of 40 each • Group A – Obese Diabetics • Group B – Non-obese Diabetics • Group C – Obese non-diabetics • Group D – Non-obese non-diabetics. Highest VF was seen in Group- A(9.16±1.93cm) Obese Diabetics. The comparison of VF between the groups was statistically significant(P- 0.000). The highest IR was also in Group- A (9.47±17.59) Obese

Diabetics. Followed by Groups B, C, and D in that order. The diabetic group, as expected, had higher IR compared to non-diabetics. BMI (30.18±5.18) and WC (101.77±10.90) were highest in Group A obese diabetics. Blood pressure, HDL, TG, and WC are part of metabolic syndrome. SBP (mmHg) 128.21±23.22, DBP (mm/Hg) 82.37±7.08, TC (mg%) 185.09±33.36, TG (mg%) 161.68±83.22, HDL (mg%) 44.24±6.65 values were higher in the Obese diabetics group(A)when compared to all other groups. SCF was high, along with a high VF in obese non-diabetics. (3.15±0.56). The author concluded that WC was a useful surrogate measure of VF. SCF and VF were found to be poor indicators of Insulin Resistance. BMI, WC, VF, and SCF did not correlate consistently with the components of metabolic syndrome, suggesting a need for reassessment of their role.

Anjana et al.[79] 2004 conducted a study on Visceral and Central Abdominal Fat and Anthropometry concerning Diabetes. 82 type 2 diabetics and 82 age- and sex-matched controls participated in this case-control research. Analysis demonstrated a reasonable degree of agreement between tertiles of visceral and central abdominal fat (=0.44, P0.0001) and a substantial association between visceral and central abdominal fat (P 0.0001).Diabetic subjects had significantly higher visceral (140.5 ± 40.6 Cm2) when compared to the nondiabetics (119.5 ± 53.5 Cm2; P=0.05). Waist circumference (87.2 ± 11.4 cm,92.3 ± 9.4 cm) and Saggital abdominal diameter (21.0 ± 2.6 cm,22.6 ± 2.5cm) showed a strong correlation with visceral and central abdominal (P <0.0001) fat in subjects with and without diabetes. BMI is higher in diabetic subjects (26.1 ± 4.2 kg/m2) when compared to nondiabetic (24.0 ± 4.7 kg/m2 Systolic blood pressure (125 ± 17 mm Hg) and Diastolic blood pressure (79 ± 11mm Hg), were more in diabetic subjects. Fasting plasma glucose (mmol/l) 9.4 ± 3.3 and HbA1c (%) 9.1 ± 2.1 were more in diabetic subjects. The study concluded that Visceral and central abdominal fat strongly correlated with type 2 diabetes.

Wang Y et al.[80] in 2005 compared the body mass index (BMI), WC, and WHR in predicting type 2 diabetes. 884 incident type 2 diabetes cases were documented. BMI and WC were positively correlated, as were BMI and WHR. A high WC was a vital risk factor for type 2 diabetes (RR: 20.4), which was much higher than that for the WHR decile (RR: 8.7) or BMI (RR: 16.5). The age-adjusted RRs across quintiles of WC at baseline were 1.0, 2.0, 2.7, 5.0, and 12.0, respectively (P for trend 0.0001). This positive association between WC and the risk of type 2 diabetes RRs across quintiles of WC were 1.0, 1.7, 2.0, 3.0, and 4.5, respectively (P for trend 0.0001). Increased WHR at baseline predicted an increased risk of

type 2 diabetes, but the association was weaker than that for WC. The age-adjusted RRs across quintiles of WHR were 1.0, 2.1, 2.7, 3.6, and 6.9 (P for trend 0.0001). Elevated BMI at baseline increases the risk of type 2 diabetes. The association was weaker for WC but more potent than for WHR. The age-adjusted RRs across BMI quintiles were 1.0, 1.1, 1.8, 2.9, and 7.9 (P for trend 0.001). The present study concluded that abdominal adiposity measured with WC and WHR is a vital risk factor for type 2 diabetes.

Ozder et al.[81] in 2014 studied the Lipid profile abnormalities seen in T2DM patients. This was a cross-sectional study, and 132 patients were recruited. The blood lipid profile results revealed that the mean values for TC, TG, HDL, and LDL in female patients were, respectively, 227.6 57.7 mg/dl, 221.6 101.1 mg/dl, 31.5 6.7 mg/dl, and 136.5 43.7 mg/dl. In male patients, the mean values for TC, TG, HDL, and LDL were 219.1 34.7 250.0 100.7 30.2 7.4 125.7 21.4 mg/dl, respectively. Diabetes patients had significantly lower mean serum levels of HDL and significantly higher mean serum levels of TC, TG, and LDL (p 0.001).FBG and TC and TG revealed a substantial positive connection (p 0.05 for each). Serum levels of TC, TG, LDL, hepatosteatosis, and HbA1c showed significant associations (P<0.05). Among all patients, in 93 (70.5%) individuals, hypercholesterolemia was found. Similarly, 117 (88.6%) people had higher LDL cholesterol levels. Only 39 (29.5%) of the subjects had average TG readings, according to the study. The study showed widespread lipid abnormalities in diabetes-triggered dyslipidemia as hypercholesterolemia, hypertriglyceridemia, increased LDL, and decreased HDL.

Khan et al.[82] in 2018 determined the homeostatic model assessment insulin resistance (HOMA-IR). In T2DM and non-diabetic participants, they examined its relationships with insulin, fasting blood glucose (FBG), and glycated haemoglobin (HbA1c). The study comprised a total of 47 T2DM patients and 38 nondiabetic controls. The level of FBG was significantly higher in diabetic patients (11.51 ± 0.55 mmol/L) compared to controls (5.19 ± 0.13 mmol/L). Diabetic patients had poor glycemic control, and their HbA1c levels were significantly higher (9.49 ± 0.31% versus 6.04 ± 0.18%) than normal subjects (Fig. 1b). Although insulin levels were comparatively higher in diabetic patients (25.25 ± 5.39 μU/mL) than controls (16.71 ± 2.02 μU/mL), this difference was not statistically significant. Diabetic patients showed a significantly high level of HOMA-IR (11.49 ± 2.10) compared to normal subjects (3.95 ± 0.46). There were highly significant correlations between HOMA-IR and Insulin in diabetic patients (R = 0.961, P = 0.001) and control subjects (R = 0.975, P =

0.001). Although HOMA-IR was not correlated with HbA1c in diabetic patients (R = -0.070, P = 0.259), it was significantly associated with HbA1c in normal subjects (R = 0.355, P = 0.029). The author concluded that Insulin resistance is highly prevalent in T2DM patients. Both FBG and HbA1c levels do not reflect the intensity of HOMA-IR in T2DM patients.

Hadaegh F et al.[83] in 2009 studied the anthropometric predictors of incident type 2 diabetes mellitus. Two thousand eight hundred one females were enrolled for the study. Over a median follow-up of 3.5 years (11 months-6.3 years), 114 individuals developed diabetes (4.1%). The mean WC in people with diabetes (95.9 ± 9.7 Cm) was higher than in nondiabetics (87.2 ± 12 cm). The mean BMI in people with diabetes (30.3 ± 4.3 kg/m2) was higher when compared to the nondiabetics (27.4 ± 5.1 kg/m2). The mean waist-hip ratio in the people with diabetes (0.61 ± 0.06) was higher when compared to the nondiabetics (0.55 ± 0.08). The mean TG was higher in people with diabetes (181 ± 1.67 mg/dl) than in people with diabetes (129 ± 1.70 m/dL). BMI, WC, WHR, and WHtR were predictive of the development of type 2 diabetes, but WHtR was a better predictor than BMI.

MATERIALS AND METHODS

Type of Study:

A hospital based cross-sectional observational study.

Study Site:

Department of Medicine, Santosh Medical College & Hospital, Ghaziabad (U.P.).

Study population:

The study population includes patients presented to the hospital's Department of Medicine, fulfilling the inclusion and exclusion criteria below.

Inclusion criteria for cases:

1. Patients with age ≥ 18 years of either sex.
2. Patients with BMI ≥ 25.0 Kg/m^2.
3. Patients with type 2 diabetes mellitus (HBA1C >6.5, FBS>126mg/dl, PPS>200mg/dl, RBS>200mg/dl).
4. Patient with written informed consent by patient/guardian.

Inclusion criteria for controls:

1. Patients with age ≥ 18 years of either sex.
2. Patients with BMI ≥ 25.0 Kg/m^2.
3. Patients without diabetes and/or a history of diabetes.
4. Patient with written informed consent by patient/guardian.

Exclusion criteria for cases and controls:

1. Patients with type 1 diabetics.
2. Patients with type 2 DM and are on glitazones and/or Insulin.
3. Presence of any acute illness.
4. Pregnant patients.
5. Patients on anti-obesity medication.
6. Comorbid conditions like pulmonary disease (COPD), HIV and TB.
7. Patients under the category of BMI < 24.9kg/m^2.

Sample Size:

In this study, we want to study the abdominal adiposity in type 2 diabetes mellitus and its correlation with components of metabolic syndrome, anthropometric parameters, and insulin resistance in type 2 diabetes mellitus. Therefore, to calculate the sample size, we need to have the estimated prevalence of type 2 diabetes mellitus and the prevalence of abdominal adiposity in type 2 diabetes mellitus patients.

To this reference, we have found that past research has reported the prevalence of type 2 diabetes in India within a range of 1.9% to 25.2%. For the sample size calculation, we had taken the upper limit of this range (i.e., 25.2%). Research work also suggested that the prevalence of abdominal adiposity in type 2 diabetes patients was 68.9%.84 On calculating the sample size using these values and the sample size formula with the correction factor by Fleiss given below:

$$n_1 = \frac{\left[\left(Z_{\frac{\alpha}{2}}\sqrt{(r+1)\bar{p}\bar{q}} + Z_{1-\beta}\sqrt{(rp_1q_1 + p_2q_2)}\right)\right]}{r(p_1 - p_2)^2}$$

And $n_2 = rn_1$

Where

n_1= number of exposed

n_2= number of unexposed

$Z_{\alpha/2}$= standard normal deviate for two-tailed test based on alpha level (relates to the confidence interval level)

$Z_1-\beta$=standard normal deviate for one-tailed test based on beta level (relates to the power level)

r= ratio of unexposed to exposed

p_1= proportion of exposed with disease and $q_1 = 1-p_1$

p_2= proportion of unexposed with disease and $q_2 = 1-p_2$

$\bar{p} = [p_1 + rp_2] / r+1$ and $\bar{q} = 1-\bar{p}$

For the Fleiss method with the correction factor, take the sample size from the uncorrected sample size formula and place into the following formula:

$$n_{1cc} = \frac{n_1}{4}\left[1 + \sqrt{1 + \frac{2(r+1)}{n_1 r|p_2 - p_1|}}\right]$$

Table 5: Sample Size: X-Sectional, Cohort, & Randomized Clinical Trials.			
Two-sided significance level(1-alpha):		95	
Power(1-beta, % chance of detecting):		80	
Ratio of sample size, Unexposed/Exposed:		1	
Percent of Unexposed with Outcome:		25	
Percent of Exposed with Outcome:		69	
Odds Ratio:		6.5	
Risk/Prevalence Ratio:		2.7	
Risk/Prevalence difference:		44	
Kelsey		**Fleiss**	**Fleiss with CC**
Sample Size - Exposed	21	20	24
Sample Size-Non-exposed	21	20	24
Total sample size:	42	40	48

Taking the sample size evaluated by Fleiss with the CC formula and adjusting it for loss to follow up as 20%, we get the final sample size of 64 patients. Hence, two groups formed were of cases (n=32) comprised of diabetic obese patients and controls (n=32) that included non-diabetic obese patients.

Study Period:

The Study was conducted for a period of one year from July 2021 to June 2022.

Method of Study:

A predesigned, self-administered proforma was designed, keeping the study's objectives at the center point. The purpose of the study was explained to the patient, and informed consent was obtained. Patients were selected for the study, which satisfies all criteria. In constructing the proforma, utmost care was taken to make it broad-based so that all the aspects desired to be studied could be incorporated into its body. Anthropometric measurements, including weight, height, WC, hip circumference (HC),W/H ratio and blood pressure, were conducted by trained staff according to standard protocols. The measurement of subcutaneous fat (SCF), pre-peritoneal fat (PPF), and Visceral fat (VF) were done by the Ultrasonologist for all patients using a GE Voluson E6 system with multiple frequency convex probe for measuring VF and linear probe for measuring abdominal wall fat.

Visceral fat thickness (defined as the distance between the anterior border of the lumbar vertebra and the posterior surface of the Rectus abdominous muscle) was measured midway between the xiphisternum and umbilicus, approximately 5 cm from the umbilicus at three positions along the horizontal line. All measurements were done at the end of quiet expiration, applying minimal pressure and not displacing or deforming the abdominal contents. Diagnostic criteria for 'Metabolic Syndrome' based on American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI). Three or more of the following: 1. waist circumference ≥102 cm for males and≥88 cm for females (for Asians,≥90 cm for males and≥80 cm for females); 2. systolic blood pressure≥130 mmHg or diastolic blood pressure≥85 mmHg or antihypertensive medication; 3. fasting plasma glucose≥5.6 mmol/L or on medication for high blood glucose; 4. HDL cholesterol <1.03 mmol/L for males and <1.30 mmol/L for females or on medications for reduced HDL cholesterol; 5. triglycerides≥1.7 mmol/L or on medications for elevated triglycerides.

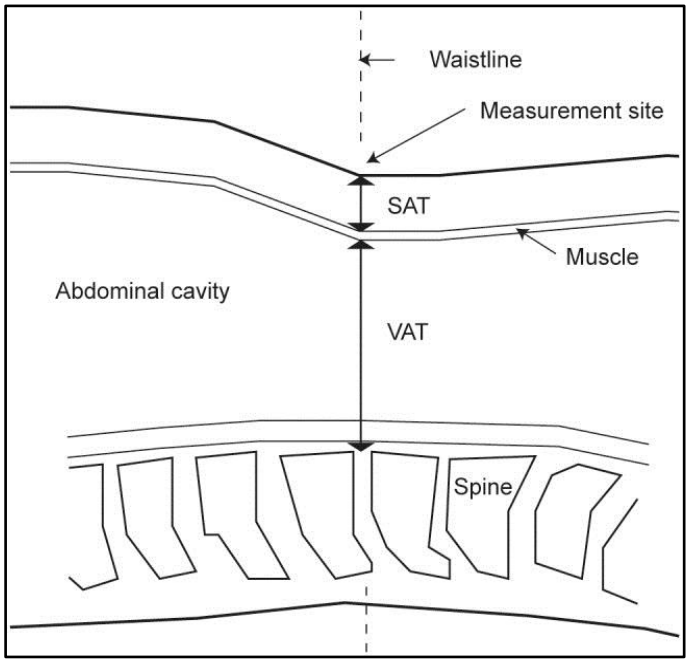
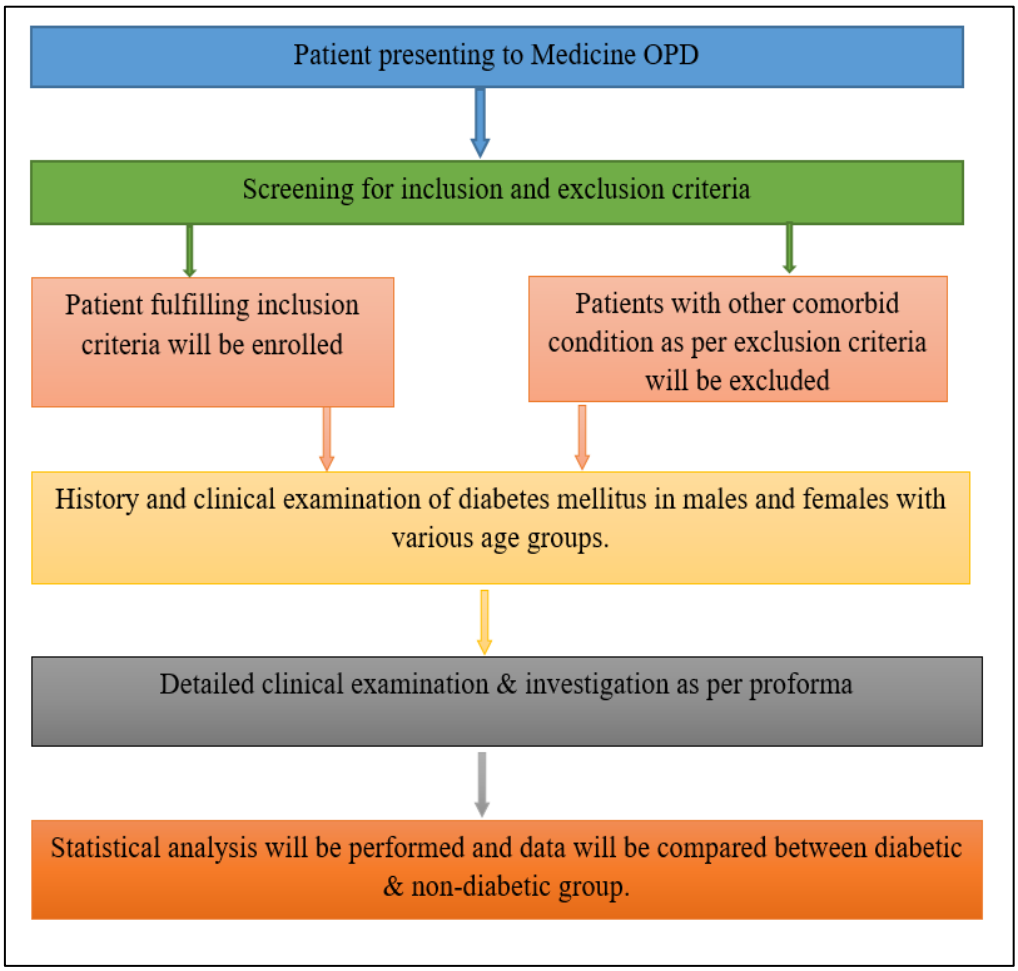


Figure 5: Measurement of visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) with ultrasonography.

Figure 6: Real time measurement of SCF and PPF by USG.

Figure 7: Real time measurement of VF by USG.

Study flowchart


Patient presenting to Medicine OPD
Screening for inclusion and exclusion criteria
Patient fulfilling inclusion criteria will be enrolled
Patients with other comorbid condition as per exclusion criteria will be excluded
History and clinical examination of diabetes mellitus in males and females with various age groups.
Detailed clinical examination & investigation as per proforma
Statistical analysis will be performed and data will be compared between diabetic & non-diabetic group.

Subject Confidentiality:

Data collection was on paper. The patients participating in the study were contacted only by the persons involved. All the patient-specific data was kept in strict confidence. Patient-identifiable data (name, contact, address, etc.) will not be presented in the journal or public forum.

Informed Consent:

The informed consent process was initiated before the individual agreed to participate in the study and continued throughout the individual's study participation. It was in a language understandable by each member of the study population. It was also submitted for ethical committee clearance.

Financial Inputs and Funding:

All the study-related procedures and investigations were done according to the institutional protocol for patient management. All these expenses were borne by the patient. There was no additional financial burden on patients due to the present research, as no other tests were

explicitly prescribed for our study. Also, this study was not sponsored by any pharmaceutical company.

Ethical Considerations:

The protocol of the present study was submitted to the Ethics Committee of Santosh Medical College & Hospital for review. After getting their approval, the study was initiated in the institution. Also, voluntary written informed consent was obtained before enrolling any patient in the survey.

Statistical Analysis:

The data was collected using a predesigned template filled by the concerned physician involved in the patient's immediate treatment. The collected data variables obtained were compiled by using an excel spreadsheet. The baseline patient characteristics are presented as frequencies for the categorical variables and as the means and standard deviations or medians and interquartile ranges for continuous variables. After appropriate data filtration, the data sheet was transferred and analyzed using Statistical Package for Social Sciences (SPSS vs. 22.0). Characteristics were compared between participants with and without diabetes at follow-up using $\chi 2$ tests.

Significant figures:

** Strongly significant (p value: $p \leq 0.01$).

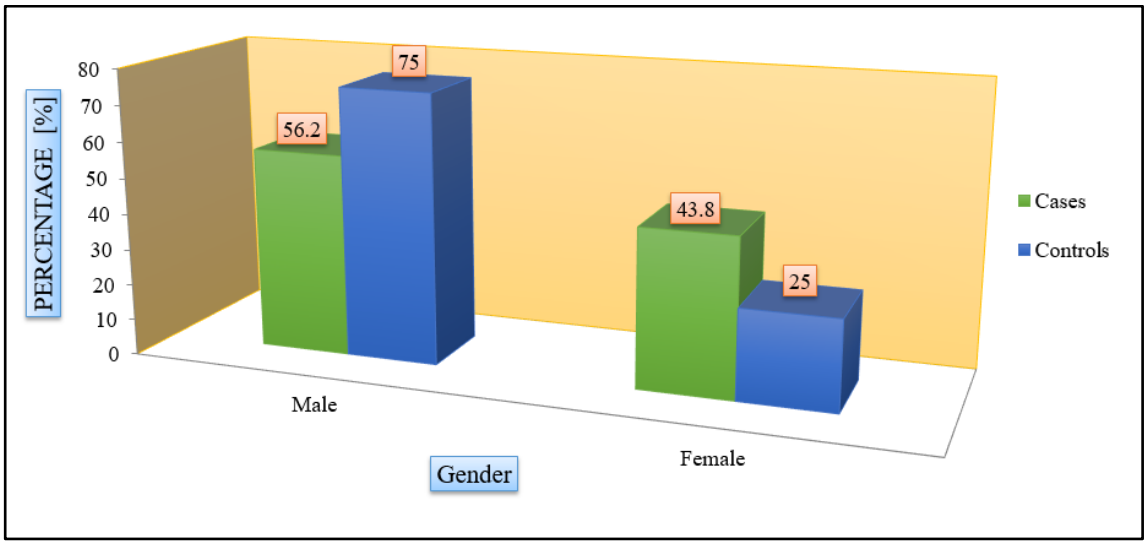
* Moderately significant (p value: $0.01 < p \leq 0.05$).

+ Suggestive significance (p value: $0.05 < p < 0.10$).

RESULTS

Table 6: Gender distribution in cases and controls.

Gender	Cases (n=32)	Controls (n=32)
Male	18 (56.2%)	24 (75.0%)
Female	14 (43.8%)	8 (25.0%)
Significance: $\chi 2=2.493$; P=0.1143.		

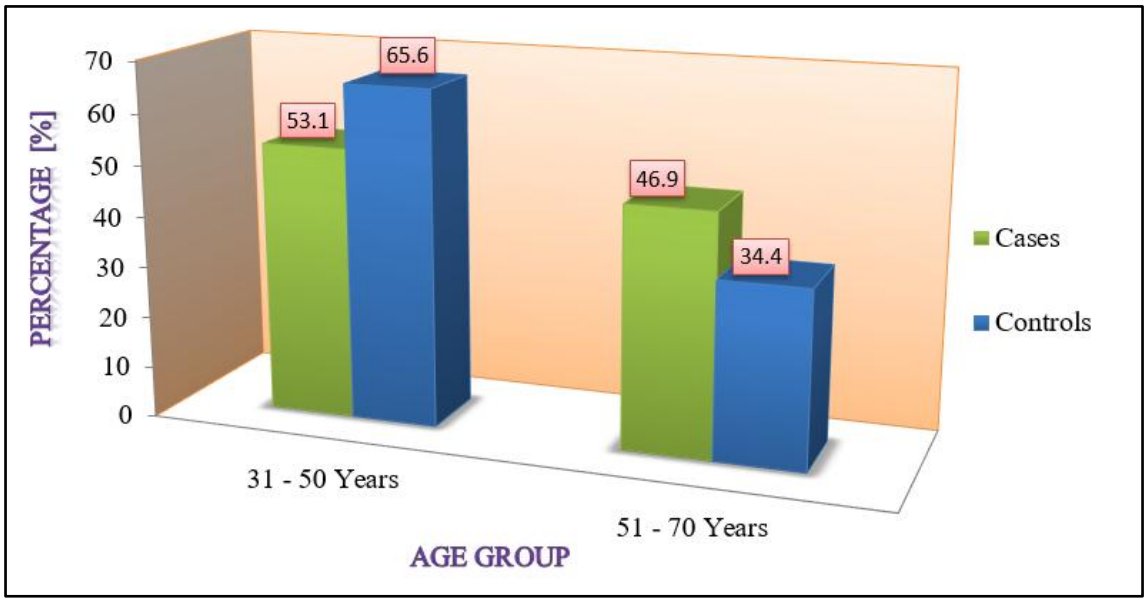


Graph 1: Distribution graph of gender in cases vs. controls.

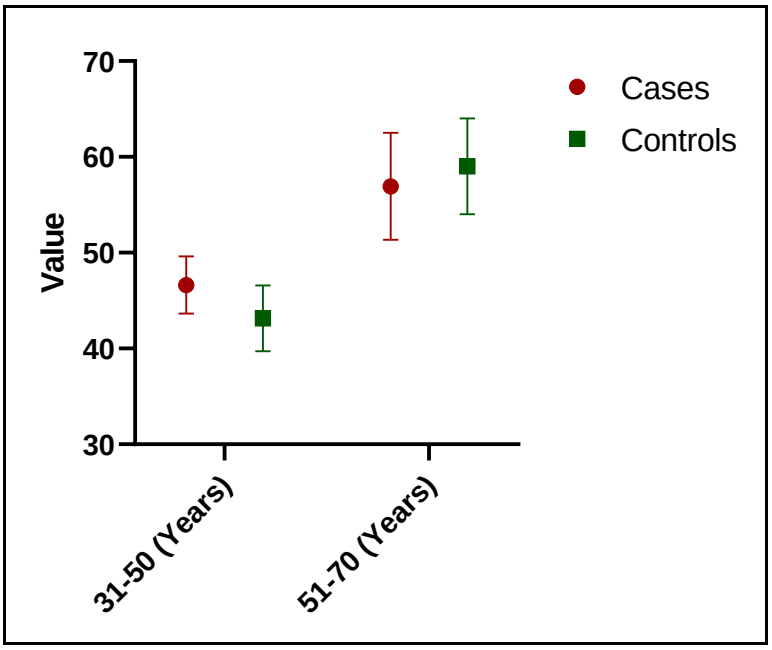
The male gender dominated both Cases and control groups. In the cases group, 56.2% were males, and 43.8% were females. While in the control group, 75% were males, and 25% were females.

Table 7: Age group distribution in cases and controls.

Age (years)	Cases (n=32)	Mean ± SD	Controls (n=32)	Mean ± SD
31 – 50	17 (53.1%)	46.63 ± 2.99	21 (65.6%)	43.14 ± 3.44
51 – 70	15 (46.9%)	56.93 ± 5.57	11 (34.4%)	59.0 ± 5.0
Significance: $\chi 2$=1.036; P=0.3086.				



Graph 2: Distribution graph of age group in cases vs. controls.

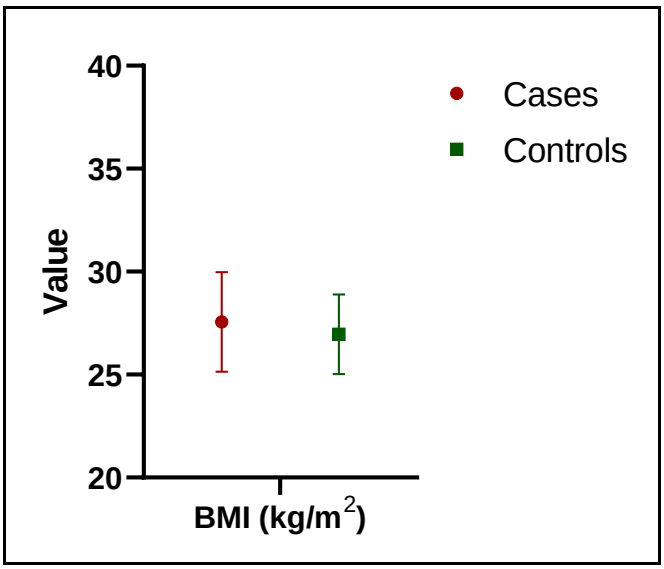


Graph 3: Mean ± SD age in cases vs. controls.

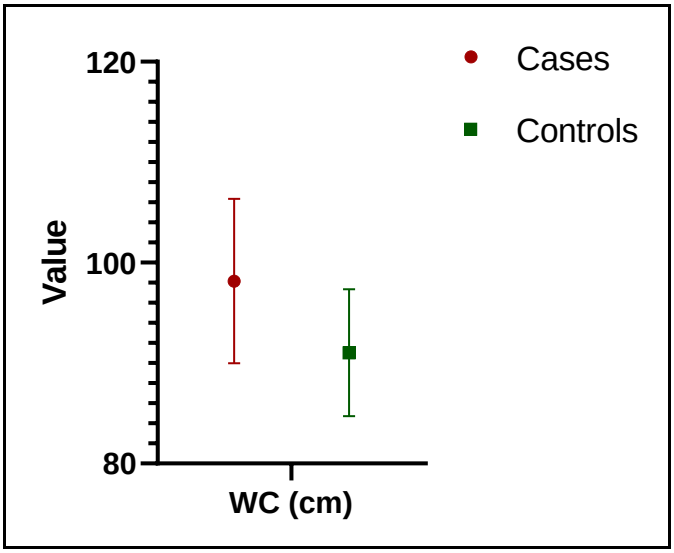
Coming to the age group distribution, both groups were dominated by patients younger than 50. In the cases group, 53.1% of the patients belong to the age group 31-50 years, and 46.9% fall in the age range of 51-70 years. Similarly, in the control group, 65.6% and 34.4% of the study participants fall in the age range of 31-50 years and 51-70 years, respectively.

Table 8: Anthropometric parameters in cases and controls.

Anthropometric parameters	Cases (n=32)	Controls (n=32)	Significance
BMI (Kg/m^2)	27.56 ± 2.41	26.96 ± 1.93	t=-1.099; P=0.2759
WC (cm)	98.16 ± 8.19	91.03 ± 6.33	t=-3.897; P=0.0002
W/H ratio	0.85 ± 0.14	0.79 ± 0.09	t=-2.039; P=0.0457



Graph 4: Mean ± SD BMI observed in cases vs. controls.

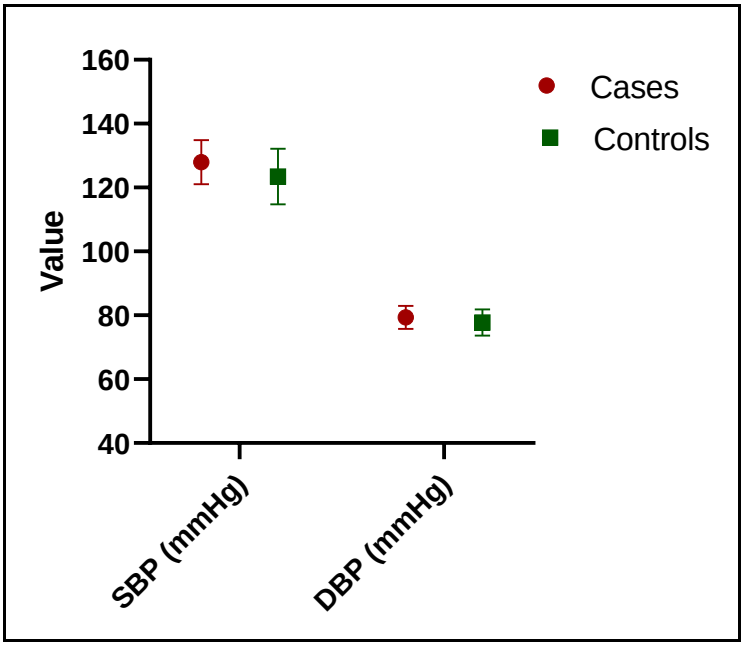


Graph 5: Mean ± SD waist circumference in cases vs. controls.

The mean BMI of the patients in the cases was found to be on the slightly higher side as compared to controls (27.56 ± 2.41 vs. 26.96 ± 1.93 kg/m2, respectively). The WC in the control group (91.03 ± 6.33 cm) was lower when compared to the cases (98.16 ± 8.19 cm). The W/H ratio of the cases and control groups was 0.85 ± 0.14 and 0.79 ± 0.09, respectively.

Table 9: Blood pressure parameters in cases and controls.

Blood pressure	Cases (n=32)	Controls (n=32)	Significance
SBP (mmHg)	127.94 ± 6.86	123.44 ± 8.70	t=-2.333; P=0.0228
DBP (mmHg)	79.35 ± 3.59	77.72 ± 4.11	t=-0.680; P=0.4993

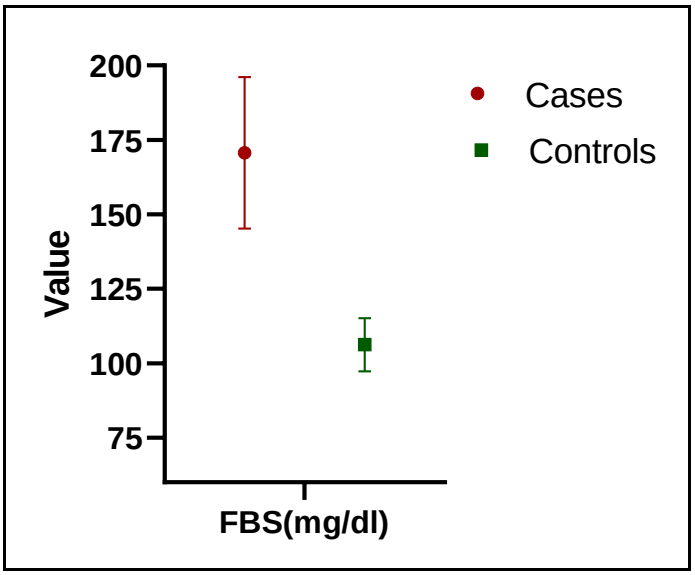


Graph 6: Mean ± SD SBP and DBP in cases vs. controls.

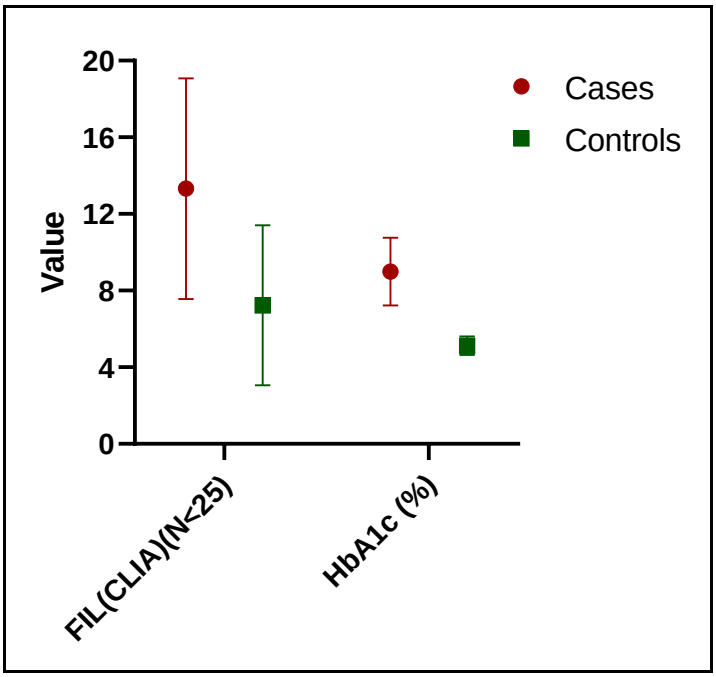
The mean SBP was slightly higher in the cases group (127.94 ± 6.86 mm Hg) when compared to the control group (123.44 ± 8.70 mm Hg). The DBP in the cases and control group was 79.35 ± 3.59 mm Hg and 77.72 ± 4.11 mm hg respectively.

Table 10: Blood sugar and insulin resistance in cases and controls.

Parameters	Cases (n=32)	Controls (n=32)	Significance
FBS (mg/dl)	170.65 ± 25.42	106.25 ± 8.92	t=-13.733; P<0.0001
FIL (CLIA)	13.32 ± 5.76	7.23 ± 4.18	t=-4.916; P<0.0001
HbA1c (%)	8.99 ± 1.76	5.12 ± 0.48	t=-12.186; P<0.0001
HOMA-IR	5.66 ± 2.51	1.84 ± 1.00	t=-8.122; P<0.0001



Graph 7: Mean ± SD fasting blood sugar in cases vs. controls.

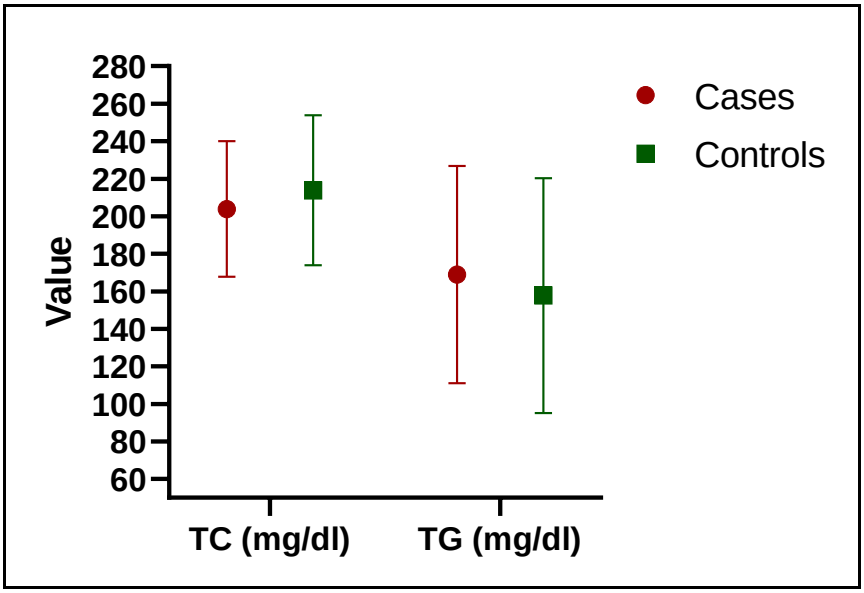


Graph 8: Mean ± SD HbA1c in cases vs. controls.

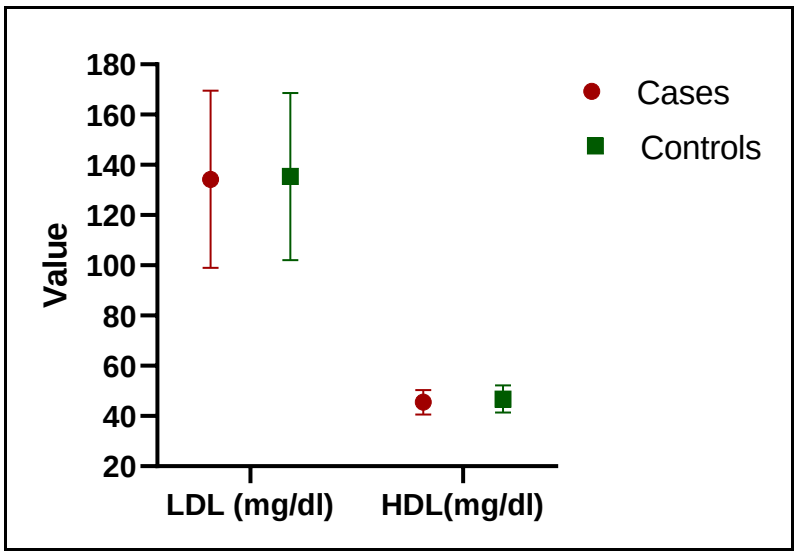
The mean FBS in the cases group was 170.65 ± 25.42 mg/dl, and the mean FBS in the control group was 106.25 ± 8.92 mg/dl. The mean HbA1c in the cases and the control group was 8.99 ± 1.76 % and 5.12 ± 0.48%, respectively. The mean HOMA-IR in the cases group was 5.66 ± 2.51, and the mean HOMA-IR in the control group was 1.84 ± 1.00. The mean FIL in the cases and the control group was 13.32 ± 5.76 and 7.23 ± 4.18, respectively.

Table 11: Lipid profile parameters in cases and controls.

Lipid Profile	Cases (n=32)	Controls (n=32)	Significance
TC (mg/dl)	203.88 ± 36.09	213.87 ± 39.99	t=-1.716; P=0.0910
TG (mg/dl)	168.94 ± 57.92	157.73 ± 62.54	t=-0.755; P=0.4527
LDL(mg/dl)	134.26 ± 35.30	135.37 ± 33.26	t=-0.131; P=0.8958
HDL(mg/dl)	45.49 ± 4.88	46.79 ± 5.43	t=1.023; P=0.3102



Graph 9: Mean ± SD of total cholesterol and triglycerides in cases vs. controls.

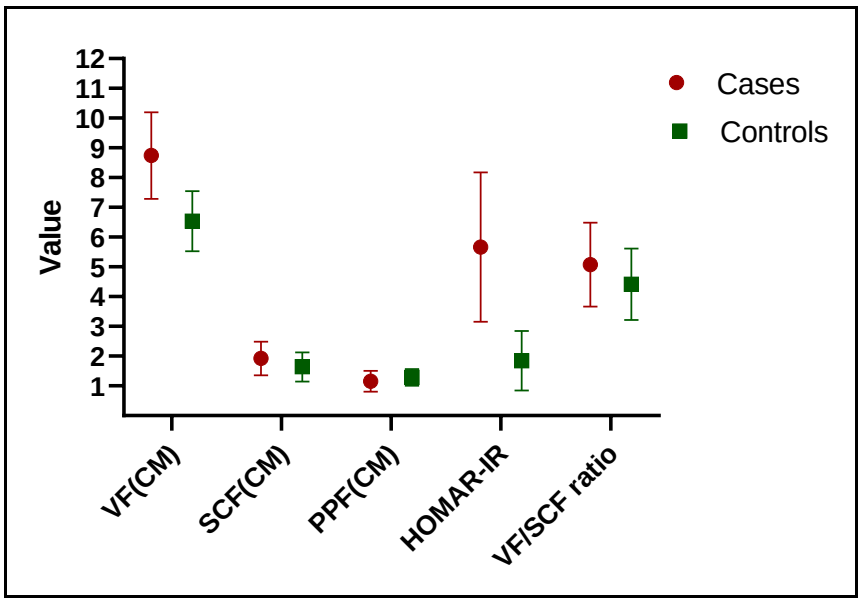


Graph 10: Mean ± SD of lipid molecules in cases vs. controls.

The mean TC in the cases and control group was 203.88 ± 36.09 m/dl and 213.87 ± 39.99 mg/dl, respectively. The mean TG in the cases group was 168.94 ± 57.92 mg/dL; in the control group, the mean TG was 157.73 ± 62.54 mg/dL. The mean LDL was almost similar in both groups, which was found to be 135.37 ± 33.26 mg/dL. Similarly, the mean HDL values were identical in both groups and found to be 46.79 ± 5.43 mg/dL.

Table 12: Body fat levels in cases and controls.

Parameters	Cases (n=32)	Controls (n=32)	Significance
VF (cm)	8.74 ± 1.46	6.53 ± 1.01	t=-7.042; P<0.0001
SCF (cm)	1.92 ± 0.56	1.64 ± 0.49	t=-2.162; P=0.0344
PPF(cm)	1.16 ± 0.35	1.28 ± 0.28	t=1.514; P=0.1350
VF/SCF ratio	5.07 ±1.41	4.41 ± 1.20	t=-2.016; P=0.0481



Graph 11: Mean ± SD of body fat levels in cases vs. controls.

The VF in the cases group was 8.74 ± 1.46 cm, and the visceral fat in the control group was 6.53 ± 1.01 cm. The SCF in the cases and control group was 1.92 ± 0.56 cm and 1.64 ± 0.49 cm, respectively. In the cases group, the PPF was 1.16 ± 0.35 cm, and PPF in the control group was 1.28 ± 0.28 cm. The VF/SCF ratio in cases and controls was 5.07 ±1.41 and 4.41 ± 1.20, respectively.

Table 13: Pearson's correlation of BMI with other parameters in cases group.

Parameter	r-value	p-value
Age	0.027	0.884 (ns)
WC	0.685	0.001**
W/H ratio	0.439	0.013*
SBP	0.065	0.728 (ns)
DBP	-0.161	0.385 (ns)
T.CHOL	0.136	0.465 (ns)
TG	0.106	0.570 (ns)
LDL	0.068	0.718 (ns)
HDL	0.157	0.398 (ns)
HBA1C	-0.372	0.039*

Parameter	r-value	p-value
FBS	0.057	0.759 (ns)
FIL(CLIA)	0.363	0.045*
HOMA-IR	0.345	0.057^{+}
SCF	0.451	0.053^{+}
PPF	-0.054	0.772 (ns)
VF	0.379	0.029*

Table 14: Pearson's correlation of waist circumference with other parameters in cases group.

Parameter	r-value	p-value
Age	-0.078	0.677 (ns)
BMI	0.685	0.001**
W/H ratio	0.292	0.110 (ns)
SBP	-0.038	0.840
DBP	-0.171	0.358
T.CHOL	0.377	0.037*
TG	0.112	0.549 (ns)
LDL	0.253	0.169 (ns)
HDL	0.090	0.631 (ns)
HBA1C	-0.179	0.334 (ns)
VF	0.425	0.017*
SCF	0.304	0.096^{+}
PPF	0.074	0.694 (ns)
FBS	-0.221	0.233 (ns)
FIL(CLIA)	0.102	0.585 (ns)
HOMA-IR	0.012	0.947 (ns)

Table15:Pearson's correlation of BMI with other parameters in controls group.

Parameter	r-value	p-value
AGE	0.087	0.636 (ns)
WC	0.368	0.038*
W/H ratio	-0.214	0.241 (ns)
SBP	0.263	0.146 (ns)
DBP	-0.236	0.193 (ns)
T.CHOL	-0.401	0.023*
TG	-0.336	0.060^{+}
LDL	-0.365	0.040*
HDL	0.250	0.168 (ns)
HBA1C	0.421	0.016*
VF	-0.017	0.928 (ns)
SCF	-0.136	0.459 (ns)
VF/SCF Ratio	0.233	0.200 (ns)
PPF	-0.140	0.445 (ns)
FBS	0.033	0.859 (ns)
FIL(CLIA)	0.172	0.345 (ns)
HOMA-IR	0.160	0.381 (ns)

Table 16: Pearson's correlation of waist circumference with other parameters in controls group.

Parameter	r-value	p-value
AGE	-0.263	0.146 (ns)
BMI	0.368	0.038*
W/H ratio	0.283	0.116 (ns)
SBP	0.258	0.154 (ns)
DBP	-0.162	0.376 (ns)
T.CHOL	0.006	0.975 (ns)
TG	-0.133	0.468 (ns)

LDL	0.023	0.901 (ns)
HDL	0.168	0.359 (ns)
HBA1C	0.213	0.242 (ns)
HOMA-IR	-0.087	0.636 (ns)
SCF	0.366	0.039*
VF	0.213	0.242 (ns)
VF/SCF Ratio	-0.238	0.189 (ns)
PPF	-0.211	0.247 (ns)
FBS	0.075	0.684 (ns)
FIL(CLIA)	-0.119	0.516 (ns)

Table 17: Pearson's correlation of insulin resistance with other parameters in cases group.

Parameter	r-value	p-value
BMI	0.345	0.057+
WC(CM)	0.012	0.947 (ns)
W/H ratio	-0.090	0.629 (ns)
HBA1C(%)	-0.201	0.278 (ns)
VF (cm)	0.121	0.516 (ns)
SCF(cm)	0.081	0.666 (ns)
VF/SCF Ratio	-0.033	0.860 (ns)
PPF(cm)	-0.228	0.218 (ns)

Table 18: Pearson's correlation of insulin resistance with other parameters in controls group.

Parameter	r-value	p-value
BMI	0.160	0.381 (ns)
WC(CM)	-0.087	0.636 (ns)
W/H ratio	0.082	0.656 (ns)
HBA1C(%)	0.456	0.009**
VF (cm)	-0.246	0.175 (ns)
SCF(cm)	-0.188	0.303 (ns)
VF/SCF Ratio	0.169	0.356 (ns)
PPF(cm)	0.013	0.946 (ns)

DISCUSSION

More than 96% of all instances of diabetes are caused by type 2 diabetes mellitus (T2DM), the most prevalent disease. Previous research has demonstrated that while abdominal obesity is linked to T2DM, Increased weight is an independent risk factor for diabetes. The present study was designed to study the association between abdominal adiposity and components of Metabolic Syndrome, Anthropometric Parameters, and Insulin Resistance in patients with type 2 diabetes mellitus. The present study was a cross-sectional observational study, and 64 patients were recruited for the study. The subjects were stratified into two groups; cases (diabetic obese patients) and the control group (nondiabetic obese patients), with each group containing 32 participants. The male dominance was (65.6%) observed in the present study compared to the females (34.4%), similar to the **Premnath et al.**[78] studies, where 60.78% were males and 39.22% were females. In the current study, the male: female ratio in the diabetic and nondiabetic groups was 1.28: 1 and 3:1, respectively. There was no significant variation in the gender distribution between the two groups (P=0.1143). **Quershi et al**.[75] reported that in both the diabetic group and the nondiabetic group, the male: female ratio was the same and it was found to be 0.3:1. **Dambal et al**.[85] stated that in the cases group, the male: female ratio was 2.8:1 and in the control group the male: female ratio was 2:1.

Most study participants were below 50 years; 53.1% were in the cases group, and 65.6% were in the control group. There was a statistically non-significant difference in the age distribution between the two groups (P=0.3086). **Daniel et al**.[86] stratified subjects into three age categories and reported that 35.1% of the study participants fall in the age range of 18-34 years; 41.1% of the study population had 35-49 years, and the rest, 23.8% were beyond the fifth decade of the life. **Chein et al.**[87] stated that 44.1% of the study participants fell in the age range of 18-44 years, 36.1% of the research participants were in the age range of 45-64 years, and the rest 19.7% had aged at least 65 years.

The identification of individuals or groups of individuals with a range of particular health criteria can be assisted by anthropometric indices used to identify obesity. No statistically significant difference in BMI between the patients (27.56 ± 2.41 Kg/m2) and the control group (26.96 ± 1.93 Kg/m2) was found in the current investigation. There was no significant difference in BMI between the two groups (t=-1.099; P=0.2759). There was a statistically significant difference in the W/H ratio in the cases (0.85 ± 0.14) group and

control (0.79 ± 0.09; P=0.0457). The difference between the WC in the patients (98.16 8.19 cm) and the control group (91.03 6.33 cm; P=0.0002) was statistically significant. Hence, in the present study, a significant difference exists in the anthropometric parameters in the cases and the control group. **Dambal et al.**[85] reported that the mean BMI of the subjects in the study group was 28.83±5.87 Kg/m2, and that of the subjects in the control group was 24.42 ± 5.5 Kg/m2. The mean waist circumference of the subjects in the study group was 92.46±10.23 cm, and that of the subjects in the control group was 88.3 ± 4.10 cm. The mean WHR of the subjects in the study group was 0.91 ± 0.11and that of the subjects in the control group was 0.61± 0.64. **Anjana M et al**.[79] reported that the mean BMI in the nondiabetic group and the diabetic group was 24.0 ± 4.7 kg/ m2 and 26.1 ± 4.27 kg/ m2, respectively and there exists a significant statistical difference between the groups (P=0.003). The WC in the nondiabetic group was 87.2 ± 11.4 cm, and in the diabetic group, the WC was 92.3 ± 9.4 cm, and the values were found to be statistically significant (P=0.002). The W/H ratio in the diabetic group was 0.93 ± 0.06, and in the nondiabetic group, the W/H ratio was 0.91 ± 0.07, and the values were found to be moderately significant (P=0.051). **Hajian-Tilaki K et al.**[88] reported that the mean BMI, WC, waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR) were significantly higher among people with diabetes in both sexes (P = 0.001).

FBS is measured by 8 h of whole night fasting. **Patel BJ et al**.[89] reported that the mean FBS level in the diabetic patient's group was higher (167.06 ± 57.24 mg/dl) than in the healthy patient's group (78.94 ± 16.70 mg/dl), and this was in line with the present study, where there FBS values were higher in the cases group (170.65 ± 25.42 mg/dl) when compared to the control group (106.25 ± 8.92 mg/dl) (P <0.0001). **Khan et al**.[82] stated that the level of FBG was significantly higher in diabetic patients (11.51 ± 0.55 mmol/L) compared to controls (5.19 ± 0.13 mmol/L). In our study, the mean Hb A1 C was higher in the cases group (8.99 ± 1.76%) when compared to the control group (5.12 ± 0.48%), and the difference was found to be statistically significant (P<0.0001). The results are incongruent with the study by **Anjana M et al.**[79], who reported that mean HBA1c in the nondiabetic group (5.6 ± 0.5%) when compared to the diabetic group (9.1 ± 2.1%). **Khan et al.**[82] reported that Diabetic patients had poor glycemic control and their HbA1c levels were significantly higher (9.49 ± 0.31% versus 6.04 ± 0.18%) than normal subjects. The mean FIL in the cases group (13.32 ± 5.76) was higher when compared to the control group (7.23 ± 4.18). **Habib et al.**[90] reported that there were significantly higher serum insulin levels in Saudi T2DM patients compared to healthy controls.

Insulin resistance (IR) is a hallmark of diabetes mellitus type 2. Several studies have shown that IR is a strong predictor for the future development of diabetes. The HOMA-IR in the cases group (5.66 ± 2.51) was higher compared to the control group (1.84 ± 1.00). There exists a tremendous significant difference in insulin resistance between the groups (P<0.0001). **Premanath et al**.[78] reported that the IR in obese diabetics, non-obese diabetics, obese nondiabetics, and non-obese nondiabetics was 9.47±17.59, 4.81±4.95, 3.88±5.49 and1.60±2.25 respectively. **Khan et al.**[82] reported that Diabetic patients showed a significantly high level of HOMA-IR (11.49 ± 2.10) compared to normal subjects (3.95 ± 0.46). **Al quarni et al**.[91] reported that the HOMA-IR was found to be significantly high in T2DM patients as compared to controls.

Due to the interdependence of carbohydrate and lipid metabolism in diabetes, various factors may influence blood lipid levels. As a result, any malfunction in the metabolism of carbohydrates leads to a disorder in the metabolism of lipids, and vice versa. A common component of the plasma lipid abnormalities seen in people with diabetes is hypertriglyceridemia, typically accompanied by reduced HDL cholesterol. Increased hepatic secretion of very low-density lipoprotein (VLDL) and delayed clearance of triglyceride-rich lipoproteins, primarily caused by increased levels of the substrates for triglyceride production, free fatty acids, and glucose, maybe the possible mechanisms causing hypertriglyceridemia. In the current study, there was no significant difference in the lipid profile (TC, TG, LDL, and HDL) in the cases and control group, respectively (P>0.05). The mean TG concentration was slightly higher in the cases group (168.94 ± 57.92 m/dl) compared to the control group (157.73 ± 62.54 mg/dl). **Ali F et al.**[92] reported that Diabetic atherosclerotic individuals (both males and females) had high levels of TC, TG, LDL, VLDL, and low levels of HDL compared to nondiabetic atherosclerotic and normal control individuals. **Ozder et al.**[93] reported that significantly higher mean serum levels of TC, TG, and LDL and significantly lower mean serum levels of HDL were noted in patients with diabetes (p<0.001). **Sharma et al.**[94] reported that the Total cholesterol was 235.18 ± 33.15 in obese diabetic patients and 152.66 ± 42.71 in obese controls. Serum triglyceride was 198.89 ± 43.10 in obese diabetic patients and 104.22 ± 18.09 in obese controls. Serum HDL-C was 36.28 ± 15.06 in obese diabetic patients and 41.08 ± 11.25 in obese controls. Serum LDL-C was 149.52 ± 31.05 in obese diabetic patients and 80.33 ± 37.22 in obese controls. The lipid profile of obese T2DM patients, when compared with obese control subjects, showed a statistically significant increase in the levels of serum total cholesterol (P< 0.001), serum

triglycerides (P< 0.001), and serum LDL-C (P< 0.001). In contrast, serum HDL-C levels did not show statistically significant variation in the two groups (P > 0.05).

In the current study, the mean VF in the cases group (8.74 ± 1.46 cm) was higher when compared to the control group (6.53 ± 1.01 cm), and the difference was found to be statistically significant (P<0.0001). A significant statistical difference existed in the SCF, with values of 1.92 ± 0.56 cm and 1.64 ± 0.49 cm in cases and controls, respectively (P=0.0344). **Anjana M et al.**[79] reported that the VF in the nondiabetic group (119.5 ± 53.5 cm 2) was less when compared to the diabetic group (140.5 ± 40.6 cm 2). While Subcutaneous abdominal fat in the nondiabetic group (208.7 ± 118.6 cm) was less compared to the diabetic group (230.1 ± 97.5 cm2). **Premanath et al**.[78] reported that the SCF and VF in obese diabetics, non-obese diabetics, obese nondiabetics, and non-obese nondiabetics were (2.79±0.72cm, 9.16±1.93cm); (2.21±0.6cm, 7.03±1.88 cm); (3.15±0.56 cm, 8.08±2.08cm) and (2.18±0.82 cm, 5.86±1.65cm) respectively. **Tayama K et al.**[95] reported that the PPF value in the diabetic males (11.1 ± 5.0 mm) was higher compared to the control (7.3 ± 0.4 mm).

BMI and WC are the two anthropometric measurements routinely utilized to grade obesity. Hence in the present research, we determined the correlation of BMI and WC with the components of Metabolic Syndrome, Anthropometric Parameters, and Insulin Resistance. BMI values in the cases group are highly correlated with WC (P=0.001; r=0.685), moderately correlated with W/H ratio (P=0.013; r=0.439), weak correlation with FIL (P=0.045; r=0.363) and VF (P=0.029; r=0.379). In comparison, BMI in the control group had a moderate correlation with HBA1C (r=0.543; P=0.007). Concerning WC values in the cases group, WC has a high correlation with BMI (r=0.685; P=0.000), moderate correlation with VF (r=0.425; P=0.017), and weak correlation with total cholesterol (r=0.377; P=0.037). WC exists in the control group; there is a weak correlation between SCF (r=0.339; P=0.113) and BMI (r=0.354; P=0.098). **Premanath et al**.[78] reported that there exists a moderate correlation between SCF in the case of obese diabetics (r=0.550) and obese nondiabetics (r=0.543); VF (r=0.552) was moderately correlated with BMI in the case of non-obese nondiabetics. While WC was highly correlated with SCF (r=0.616) in the case of obese diabetics, and WC was highly correlated with VF in the case of obese nondiabetics (r=0.681) and non-obese nondiabetics (r=0.603). Waist circumference was the best predictor of intraabdominal fat thickness in normal subjects and central obesity. **Anjana et al**.[79] reported that in the diabetic group, central abdominal fat had a moderate correlation with BMI (r=0.565) and WC

(r=0.580). Similarly, in the nondiabetic group, there exists a high correlation between Central abdominal fat, BMI (r=0.788), and WC (r=0.751). **Roopkala et al.**[96] also found that waist circumference significantly correlated with subcutaneous fat and visceral fat.

LIMITATIONS

One of the limitations of this study is that the cross-sectional studies do not allow causal relationship inferences underlying the observed associations to be drawn, and reverse causation may have played a role in our results. Secondly, the generalization of these findings to other populations may be limited as there may be variability in body composition across different populations. A significant limitation to consider when interpreting the results is the possibility of measurement error of anthropometric parameters and measurement of visceral adipose tissue and subcutaneous adipose tissue with ultrasonography which is operator-dependent. The differences in our results and other comparative studies might be due to the study design, sample selection, and differences in population, genetics, race, duration of DM, age, sex, glycemic control, and other risk factors.

CONCLUSION

The diabetic obese population possessed greater visceral and subcutaneous adiposity than controls, and the differences were more significant in visceral. In this study, BMI values in the cases group highly correlated with WC (P=0.001; r=0.685); moderately associated with the W/H ratio (P=0.013; r=0.439). Moreover, BMI was found to be a useful surrogate measure of visceral fat (P=0.029; r=0.379). Concerning WC values in the cases group, WC had a moderate correlation with VF (r=0.425; P=0.017). But, BMI, WC, VF, and SCF did not correlate consistently with the components of metabolic syndrome, suggesting a need for reassessment of their role. There was no significant correlation between abdominal adiposity and insulin resistance in either of the cases or controls. Future research could use visceral adipose tissue to subcutaneous adipose tissue fat ratios to investigate the disparity in visceral and subcutaneous adiposity distribution between diabetic and nondiabetic populations.

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ANNEXURE – I

CLINICAL PROFORMA

Particulars of the patient:

1. Name:
2. Age:
3. Sex:
4. Occupation:
5. D.O.A
6. D.O.D

History:

Presenting complaints (with duration, onset, associated factors)

1. Polyuria
2. Polyphagia
3. Polydypsia
4. Weight loss/gain
5. Nocturnal diarrhoea/constipation
6. Hypoglycaemic unawareness
7. Bladder/bowel incontinence
8. Impotence
9. Postural dizziness
10. History of significant cardiovascular / cerebrovascular disease / thyroid disorder.

Past History: History of Tuberculosis, Diabetes, CAD, COPD, Asthma, Seizures.

Family history: Special consideration to HTN, DM, TB, CAD, CVA

Personal History:

1. Dietary-
2. Sleep-
3. Bladder and bowel-
4. Smoking-
5. Alcohol-
6. Treatment History:

General Physical examination:

1. Built
2. Nutrition

3. Height(cm)/Weight(cm)/Waist(cm)
4. Waist Hip ratio:
5. BMI
6. Pallor/Icterus/Clubbing/Cyanosis/Lymphadenopathy/Oedema
7. Vitals: BP
8. Pulse
9. Respiratory Rate

Investigations:

1. Fasting Blood glucose
2. Post Prandial Blood Glucose
3. Blood Urea
4. Serum Creatinine
5. Urine routine , microscopic analysis and microalbumin
6. Lipid profile
7. HbA1c

ANNEXURE – II

CONSENT FORM

SANTOSH MEDICAL COLLEGE AND HOSPITAL, GHAZIABAD, UP

I _____S/o/D/o/W/o_____R/o_____ have been explained about the risks of this study by Dr. Jayesh Patil. I volunteer to take part in the study titled **"STUDY OF ABDOMINAL ADIPOSITY IN TYPE 2 DIABETES MELLITUS AND ITS CORRELATION WITH COMPONENTS OF METABOLIC SYNDROME, ANTHROPOMETRIC PARAMETERS AND INSULIN RESISTANCE IN TYPE 2 DIABETES MELLITUS."** and have also been explained the purpose of this study to my satisfaction. I have been informed that in case of any adverse reaction, the treatment and compensation will be the responsibility of the investigator.

My details will be kept confidential and I have the freedom to withdraw from the study at any point of time. I have been explained that I am under no risk while participating in this study.

I hereby give my consent to be enrolled in this study.

Signature of volunteer/guardian Signature of investigator

Telephone number

Signature of witness Date:

सहमति पत्र

संतोष मेडिकल कॉलेज और अस्पताल, गाजियाबाद, यू.पी.

मुझे _____S/o/D/o/W/o_____R/o_____ को डॉ. जयेश पाटिल द्वारा इस अध्ययन के जोखिमों के बारे में समझाया गया है। मैं स्वेच्छा से "STUDY OF ABDOMINAL ADIPOSITY IN TYPE 2 DIABETES MELLITUS AND ITS CORRELATION WITH COMPONENTS OF METABOLIC SYNDROME, ANTHROPOMETRIC PARAMETERS AND INSULIN RESISTANCE IN TYPE 2 DIABETES MELLITUS." शीर्षक वाले अध्ययन में भाग लेना चाहता हूं। और मेरी संतुष्टि के लिए इस अध्ययन के उद्देश्य के बारे में भी बताया गया है। मुझे सूचित किया गया है कि किसी भी प्रतिकूल प्रतिक्रिया के मामले में, उपचार और मुआवजे की जिम्मेदारी अन्वेषक की होगी।

मेरा विवरण गोपनीय रखा जाएगा और मुझे किसी भी समय अध्ययन से हटने की स्वतंत्रता है। मुझे समझाया गया है कि इस अध्ययन में भाग लेने के दौरान मुझे कोई जोखिम नहीं है।

मैं इस अध्ययन में नामांकित होने के लिए अपनी सहमति देता हूं।

स्वयंसेवी/अभिभावक के हस्ताक्षर

अन्वेषक के हस्ताक्षर

टेलीफोन नंबर

गवाह के हस्ताक्षर दिनांक:

ANNEXURE – III

PATIENT INFORMATION SHEET

1. OPD no:
2. Name:
3. Age:
4. Sex:
5. Address:
6. Phone number:

We invite you / your patient to participate in the research study titled study of being conducted in Santosh Medical College and Hospitals, Ghaziabad.

In accordance with the rules of ethical committee, a written consent will be taken. The relevant history will be asked and clinical examination of the patient will be done. Only the results of the investigations that you / your patient has already underwent as standard of care for such condition, are required for the study and no additional investigation will be done for the study. Special investigations, if any, will be sent as per the discretion of the treating physician and no additional investigation will be conducted for the purpose of the study You / your patient identity will be kept confidential throughout the study and thereafter. You are free to withdraw from the study any time without any loss of rights to undergo treatment in this hospital.

For any further questions regarding the study you can contact the investigator of study at the following number: Dr. Jayesh Patil [+91 8076828078].

रोगी सूचना पत्र

1. ओपीडी संख्या:

2. नाम:

3. आयु:

4. सेक्स:

5. पता:

6. फोन नंबर:

हम आपको/आपके रोगी को संतोष मेडिकल कॉलेज और अस्पताल, गाजियाबाद में किए जा रहे अध्ययन शीर्षक के शोध अध्ययन में भाग लेने के लिए आमंत्रित करते हैं।

नैतिक समिति के नियमों के अनुसार लिखित सहमति ली जाएगी। प्रासंगिक इतिहास पूछा जाएगा और रोगी की नैदानिक परीक्षा की जाएगी। अध्ययन के लिए केवल उन जांचों के परिणाम की आवश्यकता होती है जिन्हें आप/आपके रोगी ने ऐसी स्थिति के लिए देखभाल के मानक के रूप में पहले ही झेला है और अध्ययन के लिए कोई अतिरिक्त जांच नहीं की जाएगी। विशेष जांच, यदि कोई हो, उपचार करने वाले चिकित्सक के विवेक के अनुसार भेजी जाएगी और अध्ययन के उद्देश्य के लिए कोई अतिरिक्त जांच नहीं की जाएगी आप/आपके रोगी की पहचान पूरे अध्ययन के दौरान और उसके बाद गोपनीय रखी जाएगी। आप इस अस्पताल में इलाज कराने के अधिकार के किसी भी नुकसान के बिना किसी भी समय अध्ययन से हटने के लिए स्वतंत्र हैं।

अध्ययन के संबंध में किसी और प्रश्न के लिए आप अध्ययन के अन्वेषक से निम्नलिखित नंबर पर संपर्क कर सकते हैं: डॉ. जयेश पाटिल [+९१ ८०७६८२८०७८]।

ANNEXURE – IV

ETHICAL & SCIENTIFIC BOARD COMMITTEE CERTIFICATE

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

F. No. SU/2021/1830[18] Dated: 17.07.2021

Subject: Grant of Ethical Clearance for Thesis Protocol Topic **"STUDY OF ABDOMINAL ADIPOSITY IN TYPE 2 DIABETES MELLITUS AND ITS CORRELATION WITH COMPONENTS OF METABOLIC SYNDROME ,ANTHROPOMETRIC PARAMETERS AND INSULIN RESISTANCE IN TYPE 2 DIABETES MELLITUS."**, submitted by **Dr. Patil Jayesh Narendra,** MD/MS student of Batch 2020-2021, Department of **General Medicine.**

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"STUDY OF ABDOMINAL ADIPOSITY IN TYPE 2 DIABETES MELLITUS AND ITS CORRELATION WITH COMPONENTS OF METABOLIC SYNDROME ,ANTHROPOMETRIC PARAMETERS AND INSULIN RESISTANCE IN TYPE 2 DIABETES MELLITUS."**, **Dr. Patil Jayesh Narendra** is informed that the Thesis Protocol submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 19.03.2021, 22.03.2021, 24.03.2021, 25.03.2021, 26.03.2021, 27.03.2021 and 06.04.2021. The recommendations of the **Screening Committee** were considered in detail by the Institutional **Ethics Committee** in its meeting held on **12.07.2021** and the same was **approved** by the **Ethics Committee.**

He /She is informed accordingly for further necessary action.

[Dr. V. P. Gupta]
REGISTRAR

SANTOSH "DEEMED TO BE UNIVERSITY" REGISTRAR GHAZIABAD, NCR DELHI

Dr. Patil Jayesh Narendra
MD/MS student of batch 2020-2021
Department of General Medicine
Email: patiljayesh2208@gmail.com

Copy to:

1. The Vice Chancellor
2. The Dean, SMC&H
3. The Dean – Research
4. The Dean – Academics
5. The Director - IQAC
6. Dr Naresh kumar (Guide)
7. HOD of **General Medicine**

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Topic- STUDY OF ABDOMINAL ADIPOSITY IN TYPE 2 DIABETES MELLITUS AND ITS CORRELATION WITH COMPONENTS OF METABOLIC SYNDROME, ANTHROPOMETRIC PARAMETERS AND INSULIN RESISTANCE IN TYPE 2 DIABETES MELLITUS.

Cases

S.NO.	NAME	AGE	SEX	BMI	WC(CM)	W/H ratio	SBP/DBP	T.CHOL(mg/dl)	TG(mg/dl)	LDL(mg/dl)	HDL(mg/dl)	HBA1C(%)	VF(cm)	SCF(cm)	VF/SCF Ratio	PPF(cm)	FBS(mg/dl)	FIL(CL1A)(n<25)	HOMA-IR
1	SHAH ALAM	65	M	27.8	108	0.7	130/80	180	210	98	40	7.28	9.55	1.16	8.23	1.48	180	7	3.11
2	VIMLA DEVI	70	F	25.1	102	0.8	120/80	168	170	106	42	7.6	7.5	2.26	3.32	1.1	120	4	1.19
3	RAJ BAHADUR	48	M	25.8	101	0.8	134/80	177	105	107	49	7.54	9	1.38	6.52	1.4	204	11	5.54
4	SHAKUNTALA	50	F	26	106	0.9	126/88	155	90	88	49	10.8	8	1.4	5.71	1	114	12	3.38
5	MUKESH	58	M	26.7	98	0.8	138/80	162	126	105	40	8.2	9.4	1.7	5.53	1.24	206	12	6.1
6	BINA DEVI	51	F	26.4	102	0.7	130/80	266	314	161	52	9	7.1	1.71	4.15	1.8	188	16	7.43
7	HAKIMAN	80	M	28.7	101	0.8	128/80	182	226	80	46	7.81	9.6	2.26	4.25	1.08	208	13	6.68
8	MAYA	55	F	25.2	106	0.8	118/76	260	140	188	44	12	7.58	1.8	4.21	0.69	190	14	6.57
10	TAHIR	47	M	28	104	0.7	132/82	214	136	142	45	7.2	9	1.3	6.92	0.89	172	19	8.07
11	SHAHNAZ	53	F	27	110	0.8	140/80	211	288	176	45	8.6	8.6	1.9	4.53	1.5	180	11	4.89
12	AFSAR ALI	54	M	26.9	112	0.8	132/80	260	230	176	43	8	8.3	1.48	5.61	1.3	152	14	5.25
13	NATISHA	44	F	26.5	109	0.9	120/80	210	141	136	46	10	6.8	1.97	3.45	0.62	132	4	1.3
14	MD AKBAR	48	M	27.4	114	0.8	130/80	258	155	170	47	7.2	9.3	1.1	8.45	1.7	146	18	6.49
15	NOOR NISHA	40	F	25.9	109	0.7	120/70	158	125	86	47	8.3	8.27	1.89	4.38	0.58	168	21	8.71
16	FEROZA	50	F	25.6	108	0.8	130/80	180	200	161	52	10.1	10.8	1.63	6.63	0.82	170	22	9.23
17	OMWATI	46	F	31.2	115	0.8	124/80	260	245	168	43	8.31	7	2.6	2.69	1.3	178	16	7.03
18	RUKSANA	51	F	38	141	1	136/80	270	198	183	47	7	11.9	3.1	3.84	1.24	164	16	6.48
19	HARSH	42	M	31.4	108	0.9	128/80	170	117	99	48	7.2	9.2	2.1	4.38	0.97	178	19	8.35
20	KRISHAN KUMAR	52	M	31	112	1.1	122/80	175	109	105	48	8	9.5	1.68	5.65	0.59	188	25	11.6
21	SUNIL KUMAR	50	M	30.2	110	0.7	110/70	216	188	166	51	7.9	8.8	2.3	3.83	1.1	140	25	8.64
22	GYANESHWARI	56	F	31.6	112	1.2	120/80	211	288	176	45	8.9	7.1	1.6	4.44	0.9	180	11	4.89
23	MITHLESH SOM	55	M	30.2	115	1	132/80	171.1	152.2	81.7	59.2	9	9.5	1.9	5.00	0.7	130	8	2.57
24	WAKEELA	50	F	28.4	106	0.8	128/80	224	183	147	41	10.8	5.9	1.27	4.65	1.1	190	9	4.22
26	JAI PRAKASH	45	M	24.2	116	0.7	124/80	231	169	156	42	9.5	9.4	2.5	3.76	1.27	160	5	1.98
27	ANIL KUMAR	46	M	28.8	109	1.2	132/80	186	131	113	47	9.7	10.9	2.35	4.64	1.6	180	11	4.89
28	ANARA DEVI	60	F	28.2	112	1	130/80	176	127	96	45	8.2	10.2	1.42	7.18	1.6	168	6	2.49
29	VINOD	49	F	27.8	106	0.8	138/84	185	103	117	47	11.2	9	2.8	3.21	1.2	204	15	7.56
30	ASHA GOYAL	46	F	25.7	104	0.7	128/80	238	128	181.4	31	15.4	7.2	0.9	8.00	1.1	150	11	4.07
31	SAROJ	62	F	32.8	120	0.8	130/70	176.1	141	106.9	41	9.2	7.8	1.56	5.00	1.3	164	18	7.29
32	MITHLESH	45	F	28.1	94	0.9	136/80	180	161	146	46	8.6	7.1	2.3	3.09	1.14	182	13	5.84
33	SANJAY VERMA	55	M	28.2	114	1	120/80	210	141	140	42	10	11.7	2	5.85	1.8	204	7	3.53

Controls

S.NO.	NAME	AGE	SEX	BMI	WC(CM)	W/H ratio	SBP/DBP	T.CHOL(mg/dl)	TG(mg/dl)	LDL(mg/dl)	HDL(mg/dl)	HBA1C(%)	VF(cm)	SCF(cm)	VF/SCF Ratio	PPF(cm)	FBS	FIL(CLIA)(n<25)	HOMA-IR
1	LAKESH	60	F	28.6	116	0.9	120/70	214	136	142	45	5.2	7.5	1.7	4.41	0.95	90	6	1.33
2	MANGAL	65	M	26.3	110	0.8	130/80	175	107	104	50	4.7	9.3	1.8	5.17	1.7	100	4.4	1.08
3	PAWAN SAINI	42	M	29	120	0.9	136/80	188	107	103	48	5.3	8.4	2.5	3.36	0.92	110	6.9	1.87
4	MANIKEM	45	M	27.3	110	1	110/80	266	314	161	52	5	8.5	1.6	5.31	1.6	110	12	3.25
5	SURESH	40	M	27.8	111	0.8	120/80	175	109	105	48	4.8	9.5	1.8	5.28	1.4	110	2.7	0.73
6	SANJAY	45	M	26.5	112	0.8	130/80	214	136	142	45	5	10.2	2.3	4.43	1.36	98	3.8	0.91
7	LAXMIPRASAD	41	M	26.9	118	1	110/80	170	117	99	48	5.2	8.1	2.8	2.89	1.6	110	7	1.89
8	MAHESH	40	M	26.9	109	0.9	126/80	185	103	117	47	4.8	9.1	1.7	5.35	1.5	92	18	4.08
10	SUDESH	48	F	25.6	110	0.8	110/80	270	198	183	47	6	7	1.6	4.38	0.9	99	12	2.93
11	SHAHANA	45	F	26.4	108	0.9	120/80	158	125	86	47	4.8	7.6	1.2	6.33	0.9	100	4.4	1.08
12	MANOJ SHARMA	52	M	26.2	112	1	130/80	266	314	161	52	5.2	8.2	1.3	6.31	1	110	6.9	1.87
13	VIMLESH	62	F	25.4	106	0.7	110/70	214	136	142	45	4.5	7.3	1.8	4.06	1.7	110	12	3.25
14	MANISH	50	M	30	118	0.8	118/80	224	183	147	41	4.7	8.9	2.1	4.24	1.4	110	2.9	0.77
15	VINOD KUMAR	44	M	28.4	110	0.8	128/80	270	198	183	47	4.4	8.5	1.6	5.31	1.6	98	3.8	0.93
16	SHASHI SHARMA	47	M	27	109	0.9	110/80	214.9	149.3	133.8	51.2	4.8	7.8	1.56	5.00	1.3	110	7	1.89
17	KANTI DEVI	60	F	29.6	105	0.8	118/80	214	136	142	45	5.3	7	2.6	2.69	1.3	110	12	3.25
18	MD ASHRAF	43	M	28	110	0.7	130/70	224	183	147	41	4.8	8.4	2.5	3.36	0.92	110	2.6	0.72
19	ALOK KUMAR	47	M	25.8	116	0.8	120/80	188	107	103	48	4.6	8.8	2.3	3.83	1.1	98	3.8	0.94
20	SANJEESH VERMA	40	M	29.4	122	0.8	118/70	175	107	104	50	5.65	7.6	1.2	6.33	1.1	110	7	1.89
21	ARJUN	63	M	32	108	0.7	138/80	146	120	85	50	5.6	9.2	1.16	7.93	1.26	92	18	4.08
22	DHARAMVEER SINGH	66	M	29.7	120	0.8	130/80	169	75	99.9	54	6.4	9.2	1.8	5.11	1.1	99	12	2.93
23	PRABHA DEVI	54	F	26.4	96	0.8	118/80	192	141	116	28	5.2	9.6	1.8	5.33	1.7	100	4.4	1.08
24	BHAGCHAND GUPTA	58	M	27	125	0.9	130/80	266	188	190	36	4.8	10.7	3.1	3.45	1.5	110	6.7	1.87
25	DHIREN SHARMA	51	M	28	110	0.7	130/70	226	185	149	43	4.9	8.5	2.6	3.27	1.02	125	2.7	0.78
26	BHARAT	40	M	27.8	111	0.8	120/80	177	111	107	50	4.9	9.6	1.9	5.05	1.5	115	3.1	0.74
27	ASHFAQ BAIG	42	M	26.2	112	1	130/80	268	316	163	54	5.3	8.3	1.4	5.93	1.1	126	6.9	1.87
28	ROSHNI	58	F	25.6	110	0.8	110/80	272	200	185	49	6	7.1	1.7	4.18	1	101	12	2.93
29	MEERA	39	F	28.6	116	0.9	120/70	216	138	144	47	5.3	7.6	1.8	4.22	1.05	105	6	1.33
30	KRISHNA DUA	36	M	27	125	0.9	130/80	268	190	192	38	4.9	10.8	3.2	3.38	1.6	98	6.8	1.87
31	SHANTANU SHETH	42	M	29.4	122	0.8	118/70	177	109	106	52	5.75	7.7	1.3	5.92	1.2	116	7	1.89
32	MUKESH DHARIWAL	46	M	28.4	110	0.8	128/80	272	200	185	49	4.5	8.6	1.7	5.06	1.7	120	3.8	0.91
33	BIPIN RAVAL	44	M	29	120	0.9	136/80	190	109	105	50	5.4	8.5	2.6	3.27	1.02	108	6.9	1.87